THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

VOL. 62 AUGUST, 1949 No. 2

GRADUATE EDUCATION IN RADIOLOGY

CALDWELL LECTURE, 1948*

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AMONG the pioneer radiologists of America none deserves greater honor than Eugene Wilson Caldwell, and it is fitting that an annual tribute be paid to his memory. To be asked to deliver the Caldwell Lecture is a compliment that I appreciate deeply.

The story of Caldwell's life and work has been told many times before, but for the benefit of those whose recollections do not reach far back into the past, it may be well to recount some of the notable facts in his fine career.

Caldwell, who was born in Missouri in 1870, spent his childhood in Kansas. He entered the University of Kansas at seventeen years of age, and was graduated in electrical engineering in 1892. Shortly afterward he went to New York and became associated with the telephone company of that city. In 1898, only three years after Röntgen's discovery, Caldwell bought some simple roentgen-ray apparatus from a photographer who had found it disappointing as a source of income. From that time on Caldwell was to be a radiologist. His first application of his newly acquired equipment was as an aid to medical diagnosis and he foresaw that medicine was to be the chief field of roentgen rays. Accordingly he took a special course in anatomy, and this was followed later by a complete medical course and graduation from Columbia University and Bellevue Hospital Medical College.

Caldwell used his engineering genius and invented or designed and built much of his roentgenologic equipment, including a stereofluoroscope, a tilt table, improved types of roentgen-ray tubes and a self-regulating valve tube. He published one of the earliest papers on the roentgenologic examination of the nasal accessory sinuses and was co-author with William Allen Pusey of a book, "Roentgen Rays in Therapeutics and Diagnosis."

Appointments came to him freely, among them: director of the Edward N. Gibbs Memorial X-Ray Laboratory, visiting physician to several New York hospitals and, during World War I, major in the Medical Reserve Corps. In 1918, at the height of his renown, Eugene Caldwell, the engineer, inventor, physician and pioneer, died as a result of radiation injuries sustained in earlier years when he, like many of his

* Delivered before the Forty-ninth Annual Meeting of the American Roentgen Ray Society, Chicago, Illinois, September 14–17, 1948.

colleagues, was unaware that roentgen rays were potent, not only for good, but also for harm. In his passing the radiologic world suffered a grievous loss, a loss that was not compensated for even by the substantial legacy of advances that he left to his profession.

If Caldwell were living today he would be keenly interested in the professional education and training of radiologists, for every physician who is conscious of his obligations to the public would like his prospective associates and successors to be even more competent than himself. During most of his career, Caldwell had to acquire his knowledge of roentgen rays the hard way; that is to say, he learned chiefly by personal observation and by trial and error, for he was helping to develop a new science and art. True, he was able to pick up bits of information by visits to co-workers, by reading the scant literature and, later on, by attending meetings of small radiologic societies. But for several years roentgenology was not regarded too seriously, and what was known about it could be told quickly, for its principal use was to reveal fractures, dislocations and foreign bodies and hospital orderlies commonly acted as technicians and diagnosticians. Indeed, during the early years it was uncertain whether roentgenology was to become a trade or a profession, for it was practiced by artisans of various sorts as well as by the few physicians who sensed its possibilities.

Well after the turn of the century medical schools began to provide formal, though limited, undergraduate instruction in radiology, not as an independent field, but as a novel yet minor aid to clinical medicine. In the decade 1910 through 1919, however, advances in the diagnosis of pulmonary and gastrointestinal diseases and in radiotherapy, together with striking improvements in apparatus, increased the scope, importance and dignity of radiology so greatly that many of the medical colleges instituted organized courses of graduate training in the new specialty which now ranked with other recognized specialties. At that time and for many years after, the competence of most specialists varied widely, for each practitioner was the sole judge of his preparation and qualifications. In 1916 the ophthalmologists organized the American Board of Ophthalmology and empowered it to examine and determine the ability of candidates who sought its certificate of competence. This example was not followed by other specialists until eight years later, when the otolaryngologists established an examining and certifying board. Again there was a lull until 1930 when the process of setting up specialty boards was resumed at a rapid rate. In 1934 the American Board of Radiology was formed. In that year also the Advisory Board for Medical Specialties was instituted to advise existing specialty boards as well as groups which proposed the formation of additional boards and all those interested in graduate medical education as related to specialization. Today there are sixteen specialty boards. Their power is by no means absolute, for they heed recommendations of the Advisory Board and, in certain respects those of the Council on Medical Education and Hospitals of the American Medical Association.

It should be understood that the boards derive their prerogatives from the organized practitioners of the respective specialties and exercise limited and revocable privileges within those groups only; that the boards have neither the will nor the power to prevent the practice of any specialty by any licensed physician, regardless of his ability or lack of it; that the main purpose of the boards is to raise the standards of special practice, and that they seek to accomplish this, not by commands, but by offering a certificate of competence to all practitioners who can meet the minimal requirements and pass their examinations.

The boards have been composed of honest, fearless and conscientious men who have given freely of their time in an effort to raise the standards of their specialty without self-aggrandizement. All boards have attempted to set up their organiza-

tions in such manner as to prevent politically minded, selfish persons or groups from gaining control. If any such group ever becomes selfishly dominant, the board affected will cease to perform its proper functions.

It is obvious that specialization is a normal step in the evolution of medicine and has been largely responsible for its recent tremendous progress. It is equally obvious also that specialization has given rise to new and perplexing problems by its impact on practically every field and function of medicine. Apparently medical science, once a homogeneous unit, has disintegrated into a heterogeneous aggregation of more or less independent medical sciences and the medical sages and elder statesmen are already planning ways and means by which this seeming process of disintegration can be stopped and medicine reintegrated into a coherent and coordinated whole. We may confidently assure ourselves that all these disturbances are either purely functional or without grave organic cause, that they are natural in the sense that they are incidental to rapid growth, and that eventually they will be adjusted or will vanish of themselves.

Specialization has also profoundly affected graduate medical education. With sixteen specialty boards asking for sixteen different graduate training programs designed to fit the varied needs of all aspirants for certification, and with the medical schools overcrowded, the teaching staffs of these institutions have many difficult problems to solve. Most of these problems are outside the scope of my theme, for I shall deal only with those related to the present training program for prospective radiologists. I have chosen this subject because our program is neither perfect nor permanent but subject to change as increasing knowledge and experience warrant, and any such change can be brought about only with the advice and consent of the organized radiologists of America, including those here present, and the subject is worthy of our most productive thought, for we earnestly wish that our specialty shall rise to new heights of human service. As a basis for our considerations the requirements and recommendations set forth by the American Board of Radiology will serve excellently. In brief, the principal items are as follows:

The student in radiology must have been graduated from a medical school recognized by the Council on Medical Education and Hospitals of the American Medical Association and the American Board of Radiology and he must have completed an internship of not less than one year in a hospital approved by the Council. He must have three years of special training in radiology in clinics, hospitals or dispensaries approved by the Board and the Council as competent to give such training. During this period he shall be given graduate instruction in pathology, radiation physics and biology. Six months' full-time instruction in pathology is recommended.

The need for graduate instruction in certain of the basic sciences in the training of a prospective radiologist is obvious. There was a time when roentgen diagnosis and treatment were almost purely empirical and without any substantial underlying rationale, but today the radiologist must know the anatomic-pathologic basis of the shadows and shadow-defects that he sees, and he must have a keen comprehension of pathologic anatomy, radiobiology and radiation physics in order to devise appropriate and efficient therapeutic techniques. It is true that he is taught pathology in the early years of his undergraduate course, but the instruction is general, as it should be, and not fully remembered after graduation. What the student wants now is not only to refresh his memory of pathology in general but also to study it in its specific relationship to radiology. To most graduate students radiation physics and radiobiology will be new but deeply interesting sciences and especially valuable because of their direct and intimate relationship to radiology. These two branches also will be concerned with the broad field that has been opened by the new radioactive isotopes.

Wisely, I think, the Board has recom-

mended that six months of the three-year course be devoted exclusively to pathology. The Board does not insist on this specification, for in some institutions the department of pathology is not prepared to give six months of full-time training to all the residents in the institution, but their students can acquire a reasonable knowledge of the subject by attending all necropsies and pathologic conferences throughout their three-year residency. Training in radiation physics and radiobiology can be done satisfactorily by formal lectures and demonstrations at any time during the residency, although I feel that in the main, didactic teaching should be avoided. Not long ago I heard a renowned teacher of medicine express doubt as to the wisdom or necessity of teaching the basic sciences formally and methodically. He was strongly of the opinion that the student should "follow the rabbit"; that if, in a specific instance, he needed further information concerning a related basic science, he should have enough initiative to look up the facts in appropriate texts or should be stimulated by his instructor to do so. I agree in principle with this wise and revered teacher, but I doubt that the plan is generally applicable at present. It assumes that most students are ardent and most teachers inspiring, an ideal to be wished for and worked for but scarcely existent today. In the meantime it is to be hoped that "spoon-feeding" with formalized lectures will be held to a minimum and that a maximum of the "case system" of teaching will be applied.

Since World War II several medical schools and graduate schools have inaugurated courses in the basic sciences of six to eight months' duration. These courses were mostly didactic in nature and were conceived primarily to assist in providing training in the basic sciences for the large number of young men who were returning from military service. These courses filled a very important need by making it possible for these young men to obtain training in the basic sciences. But now that the emergency is over it is my own opinion that training in the basic sciences should and can best be given concurrently with the residency training and that wherever possible it should be given by or at the institution responsible for the residency program. Furthermore, I agree with the recent action of the American Board of Radiology whereby it will allow a maximal credit of only six months for any course or residency in the basic sciences, with the result that the student must receive a minimal training of two and one-half years in clinical radiology.

This brings us to the next requirement, which pertains to the thirty months of training in clinical radiology. The Board recommends that the student shall have active experience during this period of his training but, quite properly, it imposes no additional requirements, recommendations or even suggestions as to the division of this period among the various aspects of diagnosis and treatment or the manner in which instruction shall be given. The different institutions, unhampered by any set pattern, are free to use their differing facilities to the best advantage of the student.

In view of the opportunity offered to speak freely I want to voice some of my own ideas about the training of radiologists. At the present time the American Board of Radiology not only lends its approval to departments of radiology for the full three years of training, but also in many cases it recommends approval of certain institutions for training periods of one or two years in one or more fields of radiology. I believe that the Board should indicate specifically the phase of training for which each of these latter institutions is approved, that is, the first, second or third year. I am convinced that wherever possible the student should receive all his training at one institution or, at least, under the direction of one institution instead of dividing his three years into two or more periods in a like number of places. I am sure that the records of the American Board of Radiology indicate that those students who do spend their full three years in one place

make a much better showing than those who divide their training between two or more places.

Assuming that the resident is receiving his training in one of the institutions where the volume of work allows sufficient space, equipment and personnel to provide the ideal type of training, I believe that the three-year training program in radiology should be divided into four major periods: (1) one year in diagnostic roentgenology; (2) one year in therapeutic radiology; (3) a six-month assignment of full time in pathology, and (4) the remaining six months to be devoted to either diagnosis or therapy as the student's interests may dictate. The foregoing fulfills the minimal requirements of the American Board of Radiology and, if the student is of average intelligence, should prepare him to pass their examination.

I have found it advantageous to start the new student in one of the clinical phases of radiology and allow him to continue this phase for at least one quarter. This serves a twofold purpose: first, it allows the radiologic staff to become acquainted with him and to learn something of his interests, personality and ability, and second, it allows the student an opportunity to obtain a superficial bird's-eye view of radiology which may be of some help to him during his assignment to pathology. I also believe it is best that he receive his training in pathology fairly early in his residency. While assigned to pathology, preferably pathologic anatomy, he should be encouraged to pursue some subject of radiopathologic interest. Frequently such an effort will result in a fine piece of work suitable for a thesis which can be submitted as partial fulfillment of the requirements for an advanced degree. I realize that in small institutions it is not possible to assign all residents to six months of full time in pathology but I am sure that fairly satisfactory training in pathology can be obtained if the student is urged or even required to attend all necropsies and clinicopathologic conferences throughout his residency.

Whether the student is assigned to diagnostic roentgenology or therapeutic radiology next is of little, if any, importance. I have felt that during his service in therapeutic radiology it might be an advantage to be able to understand the roentgenograms that accompany patients who are referred for treatment, and roentgenograms that are inevitably presented in conferences which concern patients who are being treated. For that reason our students first are assigned for three months to the diagnostic section after which, if possible, they go to pathology, then to therapeutic radiology for one year and then back to diagnostic roentgenology for nine months, thereby completing the required service of one year in diagnostic roentgenology. Residents are allowed to spend the remaining six months in either diagnostic roentgenology or therapeutic radiology, whichever is preferred.

All large departments of radiology should have a radiation physicist on their staffs. Small institutions should pool their interests with similar institutions located near by and engage a competent radiation physicist to give an intensive course to their residents. It is imperative that residents in radiology receive adequate instruction in this subject. I am willing to concede that this subject can be taught satisfactorily by formal lectures, if augmented by practical demonstrations. In my institution the radiation physicist divides his instruction into three periods. During the first year approximately eighty hours of lectures and demonstrations are given. These include instruction in all phases of radiation physics. During the second year the resident is required to devote twenty to thirty hours to the calibration of machines, dosage calculations, etc. This can be done best while the student is assigned to therapeutic radiology. During the third year ten to fifteen hours of review lectures and quiz conferences are conducted.

In this connection it is noteworthy that the division of radiology into two independent specialties, diagnostic roentgenology and therapeutic radiology, is being ad-

vocated. This may occur eventually, for it is a normal, evolutionary and progressive step. At this time, however, I feel strongly that complete separation would be premature and not in the best interests of any party concerned. I will qualify this assertion with the admission that in large institutions the sections on diagnosis and therapy may well be separated physically and without interchange of personnel. But for administrative purposes both or all the sections should be under the guidance of a chairman or director, be he a diagnostician or therapist, who does not need to concern himself with the professional work of any section other than the one in which he is actively engaged. The director also should be responsible for the training program, which should continue to provide instruction in all phases of radiology, including the radioactive isotopes. This unity in training still is desirable for all new radiologists and indispensable for those who associate themselves with small groups or hospitals or engage in individual practice; in all of which situations the complete division of radiology is now impracticable.

Likewise in the larger institutions, further specialization within the fields of radiology by the teaching staff is of advantage not only to the patients whom they serve but also to the young men whom they train. In such institutions it is natural that each member of the teaching staff should seek to excel in some one phase of radiology within the field of diagnosis or therapy. I have always encouraged this practice among my own colleagues and am of the opinion that it should be encouraged generally, as it undoubtedly results in better routine work and more efficient training.

It is important to note that while the Board carefully refrains from prescribing methods of instruction, it does say that the student shall have "an active experience," by means of a residency during his two and one-half years of clinical training. The word "experience," used wisely, I think, implies actual participation in radiologic work, with a minimum of lectures like those with which he was surfeited during his undergraduate course. It implies that as the student progressively acquires the ability to execute individual details of diagnosis or therapy he shall be entrusted with those details and eventually shall be given tentative and closely supervised responsibility for the diagnosis or treatment of patients assigned to him. So valuable is this sort of experience that superior students should, if possible, be given an opportunity to serve temporarily on the staff of the department as first assistants for at least six months, or preferably a year, after completion of a three-year residency. Several of our residents after having served in this capacity, have told me that they considered this extra year to be the most valuable period of their entire training.

Occasionally a student becomes intensely interested in some single phase of radiology. For example, during the past year one of the residents under my supervision asked permission to be assigned for three months to neurologic pathology and another requested six months' assignment to radioactive isotopes. Both requests were granted but the time spent on these assignments will not be credited as a part of the required three-year residency but as additional training time. A student may, during his second or third year, decide that he wishes to devote his full time to either diagnostic roentgenology or therapeutic radiology. I recently had occasion to counsel one of our residents who had elected to limit his practice to therapeutic radiology. I advised this young man to take the minimum of training in diagnostic roentgenology and to devote the remainder of his allotted time in clinical radiology to therapeutic radiology. In addition, I advised him to request an extension of his residency for another year, during which time he should ask for training in allied clinical and surgical fields, such as gynecology, urology, etc.

I am told, unofficially, that there are now one or two large institutions in this country that have added a fourth year to their residency in radiology. Whether the additional year in these instances is compulsory

or elective, I do not know, but if it is the latter I hasten to congratulate them for the reasons which I have mentioned.

For many years it has been my practice to schedule a minimum of formal lectures and a maximum of semiformal seminars and informal conferences. The latter have proved to be efficacious and acceptable methods of instruction. Commonly the seminars, held weekly except during the summer, are devoted to a single subject which has been assigned to and prepared by one of the students serving the second or third year of his residency. He may prepare a paper although he is not allowed to read it, but must present his discussion from notes and lantern slides. Members of the staff attend and participate in the discussions and questions put to the resident who conducts the seminar. Subjects which deal with both diagnosis and treatment are presented during the year and at times residents or staff members from related clinical and surgical fields are invited to conduct one or more of the weekly seminars.

As mentioned previously, many informal conferences are held. These conferences are attended by the residents and staff members of one of the sections; for example, the Section on Therapeutic Radiology conducts what Dr. Leddy calls "the Friday night informal, unceremonious gathering" of all residents assigned to therapeutic radiology as well as the staff members of the section. These meetings are held at the home of one of the residents. Two or more subjects related to therapeutic radiology are discussed freely and informally during which time many provocative questions are asked. Similarly, in the diagnostic section informal conferences are held once or twice weekly and they deal with interesting cases encountered, and are based on roentgenograms and other data selected from material considered at film-reading periods during the day. To these data may be added the clinical history and findings at operation or necropsy. Discussion is informal and usually thorough. Residents are also encouraged to write papers on radiologic subjects to be presented and discussed at extra seminars or, occasionally, at the general staff meetings of the institution. The preparation of papers is a valuable method of self-instruction, for the writer invariably learns much from his effort whether his audience is edified or not.

Our residents, during their assignment to the diagnostic section, are expected to attend all film readings and are encouraged to discuss or even question the diagnosis or opinion rendered by the staff member. This not only serves to keep the students alert but also the staff members as well. Another important adjunct to informal teaching is a common loafing room in the department where the residents and staff members may gather during their idle moments and discuss the many problems that plague our world today. Invariably, within a few minutes after a member of the teaching staff joins the group, a subject concerning radiology or some allied field will come up for discussion or argument. Usually both the staff members and the students assembled benefit by such informal debates.

Throughout his residency the student should constantly refresh and extend his knowledge of clinical medicine in general, for he is preparing himself to be not merely a specialist but a medical consultant with a primary, but not exclusive, interest in radiology. As such he will often be required to confer with his colleagues in other fields and he should be able to contribute intelligently to the complete correlation of all the data in any case at hand. Certainly radiologists, with their domain overlapping almost every other field, should be the last to furnish justification for the sarcastic witticism that a specialist is one who knows more and more about less and less. In my institution, like all large and many small ones, weekly teaching conferences are held in the various medical and surgical sections. These conferences are open to the staff and residents of that and related sections. An example is the weekly diagnostic chest conference conducted by the medical chest service but the thoracic

surgeons, radiologists, pathologists, bronchoscopists and their residents are permitted to attend. Similar conferences of interest to residents in radiology are conducted by the sections on gastroenterology, urology, neurology, pediatrics, etc. In addition, weekly clinicopathologic conferences and staff meetings are held which are open to the entire staff and residents of the institution. I realize that it is impossible for a resident or anyone else connected with a large teaching medical organization to attend all the meetings, seminars and conferences that are scheduled but by attending as many of these extraradiologic conferences as possible the future radiologist will broaden his knowledge of medicine and surgery and thereby be a better radiologists.

Today there is no paucity of applicants for graduate training in radiology. In most institutions there are from five to twenty applicants for each appointment. Therefore, extreme care should be exercised in the selection of students and only the most promising should be accepted. It is not always an easy task to select the most promising student, for there are several factors to be considered. First among these I would place the student's ability as indicated by his grades in academic and medical schools and by his ability to apply what he knows. It is difficult to determine exactly what scholastic standing one should demand of a prospective resident, but I always feel safe if he ranks in the upper third of his class in medical school. It has been said that the average intelligence quotient of our population is 100, and it is estimated that those in the upper third of their medical class have an average intelligence quotient of 125 to 135, which places them in the upper 5 per cent of the intelligence level of the population. I would next consider the interest or drive of the prospective student. Why is he interested in radiology and what are his aims after completion of his training? Last, I would check carefully his personal characteristics, such as his ability to get along and work with his fellow man, his personal habits, intellectual honesty, etc. Although a personal interview with the applicant is often of great help, much of the information in regard to his personal characteristics must be obtained from personal references that he furnishes and are known to me. If all of us will exercise extreme care in the selection of those whom we are to train, I am sure we need not worry about the future of radiology.

These are some of the facts learned and some of the opinions formed by one man from many years of experience as a teacher of radiology and member of the American Board of Radiology. Even so, nothing said here can be considered authoritative, for neither medicine nor any of its divisions recognizes any authority except a broad consensus of its practitioners. It would be folly for me or anyone else to attempt to lay down a pattern of graduate instruction in radiology that would be applicable to all institutions, whether large or small. Even in the large institutions the method and sequence of instruction might vary considerably without making the training any the less effective. Furthermore, I wish to declare emphatically that neither the size of the department of radiology nor of the institution which it serves can be taken as an index by which to measure the quality of training. Some of the best training I know is given in departments of radiology with a teaching staff of one man. In such cases the chief is usually a strong, dynamic character who has the ability to inspire his students.

In view of what Eugene Caldwell was able to accomplish without benefit of planned training or guidance, what a grand opportunity for advancement and service is presented to the young student of radiology today!

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Vol. 62, No. 2

CONGENITAL OBSTRUCTIONS OF THE RESPIRATORY TRACT*

I. TRACHEAL MALFORMATIONS

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NOISY breathing observed in infants by their mothers or pediatricians is a common indication for roentgenologic studies. It seems normal or at least usual for infants to make some noise in breathing on occasion. When this becomes abnormal in character and an indication of respiratory obstruction is often a point requiring careful and experienced judgment. The frequent association of obstruction of various types in the pharynx, larynx, trachea and bronchi with respiratory infections is generally appreciated. In the past the role of the thymus in producing respiratory obstruction in infants has been emphasized and perhaps in some instances overemphasized. Recently attention has been called to vascular malformations which may produce compression of the trachea. It is the experience in this hospital that anomalies of the great vessels, even when vascular rings are present around the trachea and esophagus, usually do not produce significant compression or narrowing of these structures. Abnormal masses of various types in the neck or mediastinum may produce a similar compression and obstruction. Two factors are present to influence the degree of obstruction—the extrinsic force and the ability of the trachea or bronchi to resist compression. Sgalitzer[12] has emphasized this consideration in adults and it would appear to have a particular applicability to infants in determining the obstructive effects of anomalies of the great vessels, abnormal cervical and mediastinal masses, and the large thymus. It is the purpose of this communication to consider intrinsic factors of the trachea causing respiratory obstruction in infancy either alone or in combination with extrinsic factors.

CASE I. R. D. This infant was admitted to the hospital at the age of two months with the history of heavy breathing and cough since birth. A brother had died of cystic fibrosis of the pancreas and that diagnosis was made in this case. A narrow air passage in the trachea was observed and the possibilities of a congenital stenosis or a reduction in the airway by thick mucus along the tracheal wall were entertained (Fig. 1). The infant died at ten weeks of age and a cystic fibrosis of the pancreas was found and also a very narrow trachea. An indentation was seen along the anterior surface corresponding with a ridge or spine projecting into the lumen which gave off the cartilaginous rings like asymmetrically placed ribs.

CASE II. S. O. This infant was born in February, 1944, and was first seen at three weeks of age with the story that he had had a cold, cough and nasal discharge since birth. At one month he was observed to be in respiratory distress and there was thought to be difficulty in the passage of air through the trachea. During the next three years there were eleven admissions to the hospital for severe respiratory distress precipitated by relatively mild respiratory infections. Bronchoscopic studies were not conclusive and adrenalin was without effect. Lipiodol studies of the tracheobronchial tree at two years eight months of age demonstrated a narrowing of the trachea above the bifurcation and an anomalous origin of the right upper lobe bronchus (Fig. 2). This is also shown in the autopsy photographs. The bronchi were of larger caliber than the lower trachea and the cartilaginous rings in the trachea were found to encircle the trachea completely with an absence of the usual membranous portion in the

* From the Children's Hospital of Michigan, Detroit, Michigan. Presented in part at the Forty-ninth Annual Meeting, American Roentgen Ray Society, Chicago, Ill., Sept. 14–17, 1948.

posterior third of the tracheal wall. A cleared specimen of the opened trachea further shows an irregular formation of the tracheal cartilages above the bifurcation. There was no cardiovascular abnormality and the thymus weighed only 7.5 gm.

Atresia of the trachea is extremely rare and is of course incompatible with life. Marek[8] in 1940 reported such a case and quoted similar cases reported by Fritz (1933) and Payne (1900). In Marek's case the trachea ended as a blind sac just below the larynx. The esophagus was normal except that 3 cm. above the diaphragm there was a small orifice opening into both main bronchi. The much more common tracheo-esophageal malformation is esophageal atresia with tracheo-esophageal fistula. Gruenwald[2] explains this by the theory that the tracheal segment grows more rapidly than the esophagus and than when there is an incomplete tracheo-esophageal differentiation the esophageal segment is drawn out so that only esophageal remnants are found in the dorsal tracheal wall.

Congenital stenosis of the trachea usually with a malformation of the tracheal cartilages is also considered a rare anomaly. Montandon[9] reported a case in 1944 and reviewed 11 cases previously reported in the European literature. In his case there was a tracheal stenosis 1 cm. below the vocal cords requiring tracheotomy at eleven years of age. The diameter of the stenotic area measured 6 mm. compared with a measurement of 16 mm. in the lower normal portion of the trachea. Only 4 of the reported cases survived infancy. The stenosis may be generalized (Sankott,[10] Wolman[13]), or localized (Haardt,[4] Montandon[9]). It may be associated with complete cartilaginous tracheal rings (Sankott,[10] Gabriel and Feyrter,[1] Scheid,[11] Wolman[13]), or with hypoplasia of the tracheal cartilages (Guisez[3]). Variations may occur in the situation, number and disposition of the

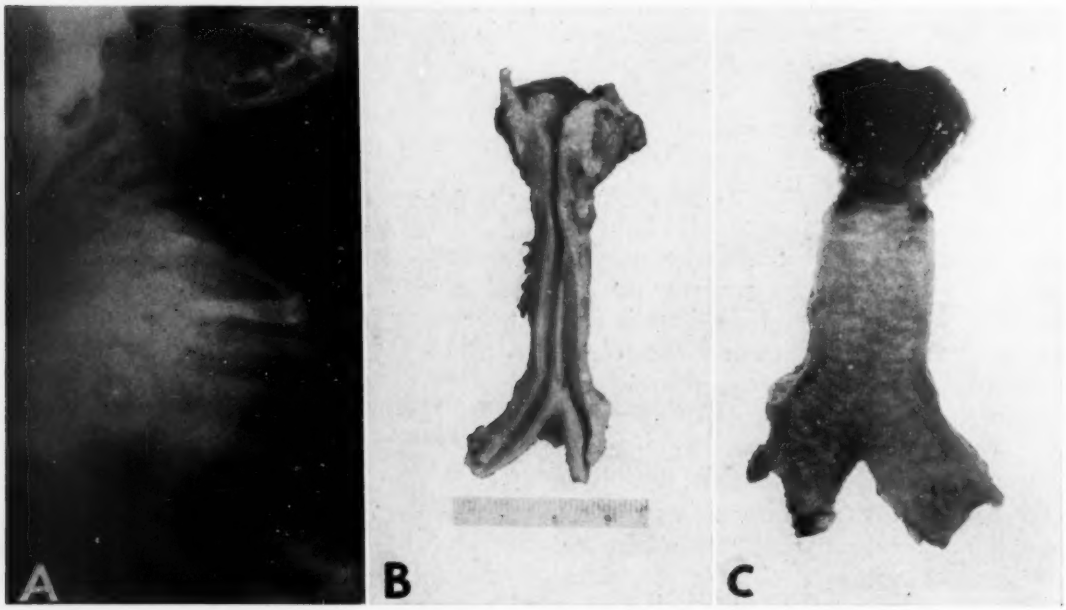

Fig. 1. Case 1. *A*, marked narrowing of the air passage of the thoracic portion of the trachea in its antero-posterior diameter observed in two month old infant with dyspnea and cough since birth. *B*, postmortem specimen of trachea opened posteriorly showing the narrow tracheal lumen and a ridge projecting from anterior surface. *C*, trachea after clearing in glycerin, opened and flattened between glass slides showing bizarre formation of tracheal and bronchial cartilages.

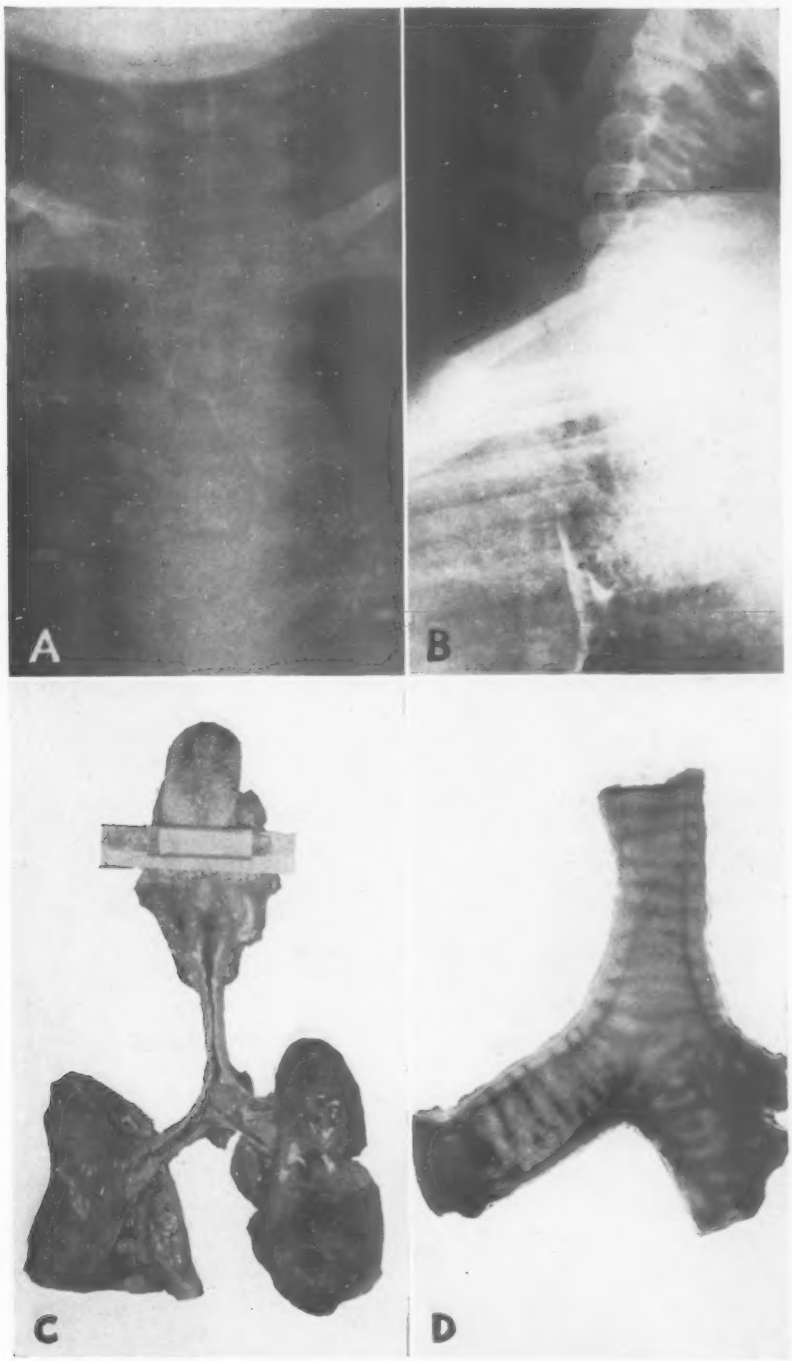

Fig. 2. Case II. *A* and *B*, anteroposterior and lateral views of the trachea in a two year eight month old infant who had suffered repeated attacks of severe dyspnea, precipitated by mild respiratory infections. The tracheal lumen is narrow particularly above the bifurcation and there is an anomalous origin of the bronchus to the right upper lobe. *C*, postmortem specimens of trachea and bronchi opened posteriorly showing the lumen of the trachea smaller than that of the bronchi. *D*, cleared and pressed tracheobronchial tree. The tracheal rings completely encircle the trachea. The rings are irregular and there is narrowing of the tracheal lumen particularly above the bifurcation. The anomalous origin of the right upper lobe bronchus is again demonstrated.

cartilaginous rings without stenosis (Gabriel and Feyrter[1]) and it is of interest that closed tracheal rings normally occur in most birds, in a few reptiles, and in some apes and humans (Sankott[10]). The narrow trachea was visualized roentgenographically in the cases of Haardt[4] and Wolman.[13]

These 2 cases reported above were seen in the Children's Hospital of Michigan in the same year and 3 similar cases have been seen, 1 of which died without autopsy and 2 are still living, therefore without pathological proof of the diagnosis.

Perhaps this condition would not be regarded as extremely rare if the trachea were examined more carefully in routine roentgen studies of the infantile chest and if the pathological picture were not overshadowed by the complicating respiratory infection which would seem to be the common precipitating cause of death.

It is usually possible to obtain a satisfactory visualization of the air passages in the pharynx and trachea in lateral roentgenograms of the infant chest (Fig. 3). Usually there is very little or no alteration in the caliber of the infantile trachea during respiration but it is not uncommon to observe variable degrees of narrowing of the trachea in its anteroposterior diameter if the exposure is made in a phase of expiration (Fig. 4). The variable behavior of the trachea in infants suggests a variable rigidity of the tracheal cartilages which would tend to disappear as the tracheal cartilages become more solidly formed. The common observation of buckling of the infantile trachea at the cervical thoracic junction is another indication of the softness and pliability of the infantile trachea. Thus the single observation of a narrow trachea is not sufficient evidence for a diagnosis of tracheal stenosis.

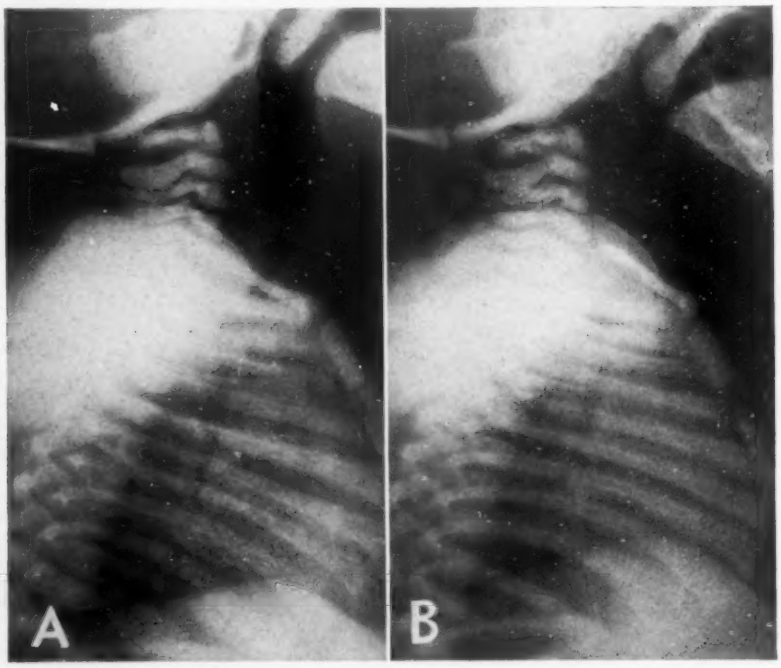

Fig. 3. Appearance of the normal infantile trachea at five weeks of age. Exposures made in inspiration (*A*) and expiration (*B*) demonstrate a constant caliber of the air passage in the trachea.

Transient narrowing of the trachea in serial studies is occasionally observed. The studies in Figure 5 are from a two to three weeks old infant with wheezing spells and a harsh non-productive cough. There was

a right upper lobe bronchopneumonia with an uneventful recovery.

We have considered, as possible explanations for transient narrowing of the trachea, spasm which seems unlikely in view of the paucity of muscular tissue in the tracheal wall, mucus overlying the tracheal mucosa, and soft tracheal cartilages. This latter possibility seems the most probable explanation in most instances.

Transient tracheal narrowing may also occur where external influences are in evidence, probably in addition to soft tracheal rings. The studies in Figure 6 are from an infant who had a tracheo-esophageal fistula which was repaired. The upper esophageal segment was much larger than the lower and there was some postoperative stenosis at the site of anastomosis. In this situation the upper segment tended to dilate with gas or food and when this occurred the adjacent trachea was narrowed in its anteroposterior diameter.

Narrowing of the trachea may be seen as the result of pressure anteriorly by a large thymus. The studies in Figure 7 are from a patient who was examined at six weeks of age for noisy breathing, cough and nasal discharge. The large thymus and narrow trachea were recognized but little significance was attached to this observation by the clinicians. Noisy breathing was again noted at two subsequent visits and at six months of age the baby was brought back with a very noisy rattling respiration which was aggravated by an upper respiratory infection. The mother gave the story that the baby's respirations were never quiet and became much more noisy with the appearance of a "cold." The large thymus and narrow trachea were again demonstrated and the decision was made to irradiate the thymus with the

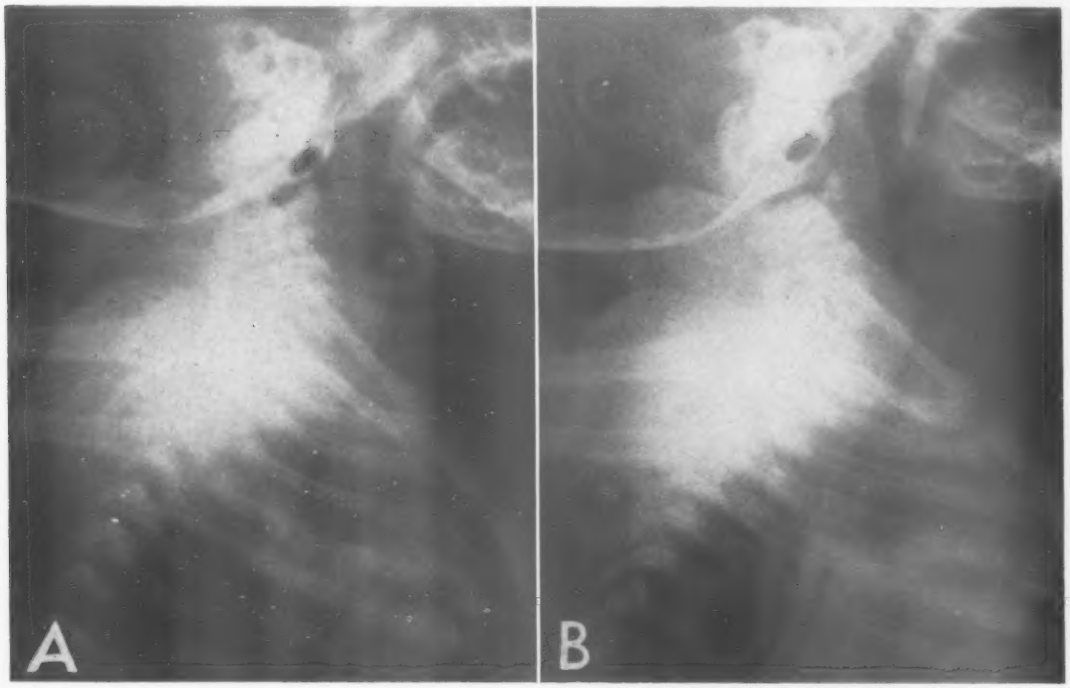

Fig. 4, *A* and *B*. Appearance of a normal infantile trachea at five weeks of age demonstrating a slight variation in the caliber of the air passage in the thoracic portion of the trachea at different periods in the respiratory cycle.

roentgen ray. The treatment consisted of three exposures of 140 r at weekly intervals, distance 25 cm., 115 kv., and 5 ma. A

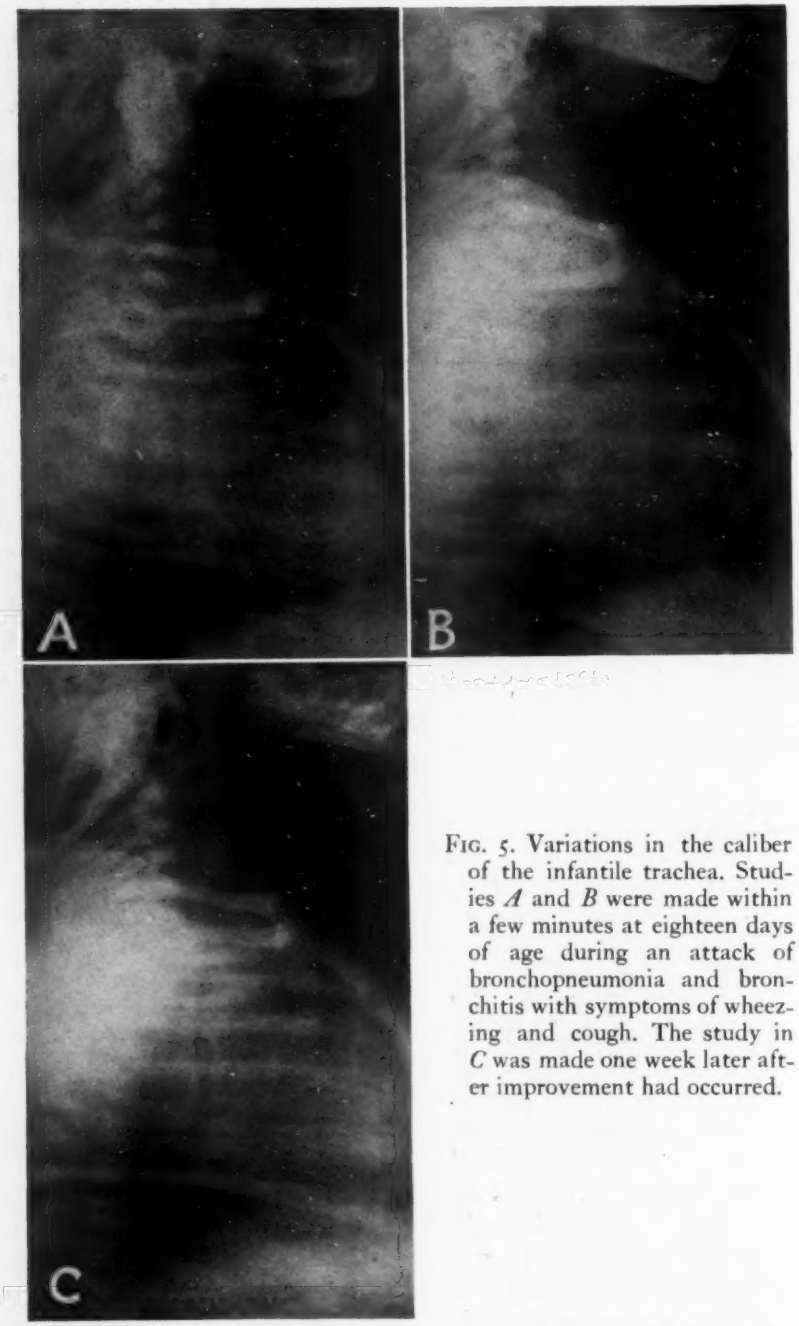

Fig. 5. Variations in the caliber of the infantile trachea. Studies *A* and *B* were made within a few minutes at eighteen days of age during an attack of bronchopneumonia and bronchitis with symptoms of wheezing and cough. The study in *C* was made one week later after improvement had occurred.

month later by the mother's account and our observations the breathing had become remarkably silent. The thymus had become inconspicuous and the trachea was of larger caliber although it had not yet attained to full size. Jackson[5,6] and other experienced bronchoscopists have testified to the fact that a large thymus may cause compression and stenosis of the trachea. It is admittedly difficult to provide autopsy proof for those who are skeptical of this

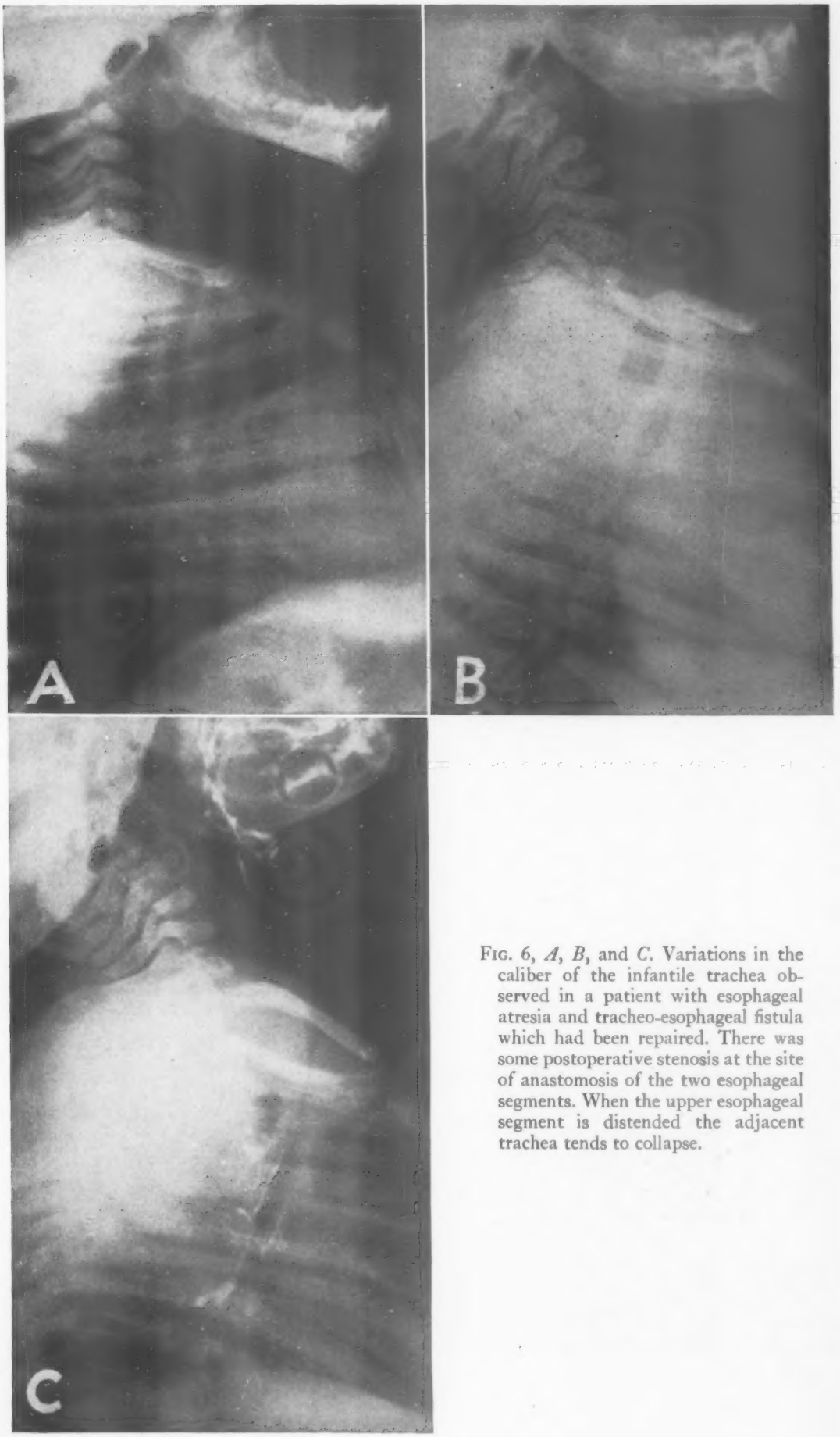

FIG. 6, *A*, *B*, and *C*. Variations in the caliber of the infantile trachea observed in a patient with esophageal atresia and tracheo-esophageal fistula which had been repaired. There was some postoperative stenosis at the site of anastomosis of the two esophageal segments. When the upper esophageal segment is distended the adjacent trachea tends to collapse.

interpretation of events but such a case has been reported by Lange[7] in an article published in 1898 entitled "Sudden Death as the Result of Compression of the Trachea by an Enlarged Thymus." A baby died suddenly at three and a half months of age in cyanosis. The trachea was narrowed about 3 cm. above the bifurcation to one-third of its normal caliber and the membranous portion of the trachea was abnormally broad in the area of narrowing. The thymus measured 7 by 4 by 3 cm., weighed 22.5 gm., and almost completely encircled the trachea in the area of narrowing. No other cause of death was found.

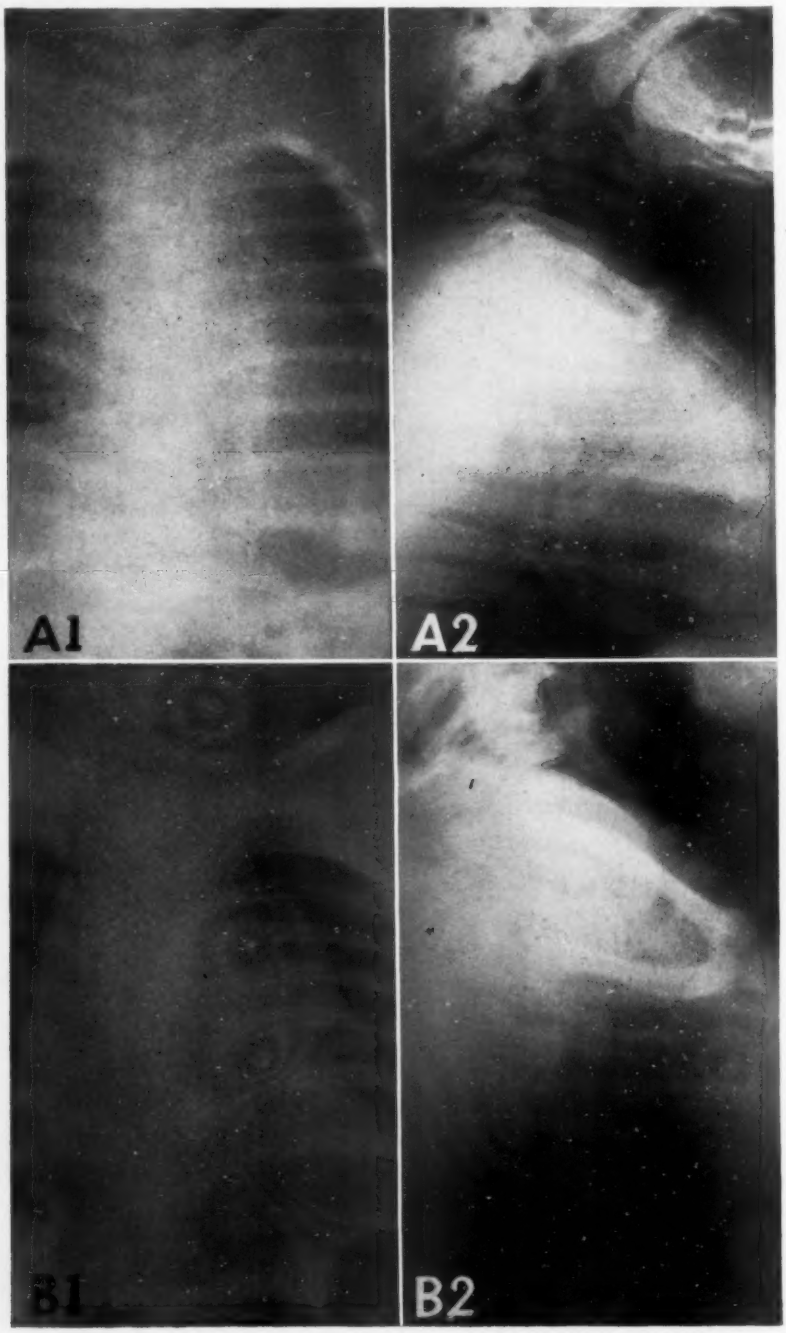

Fig. 7

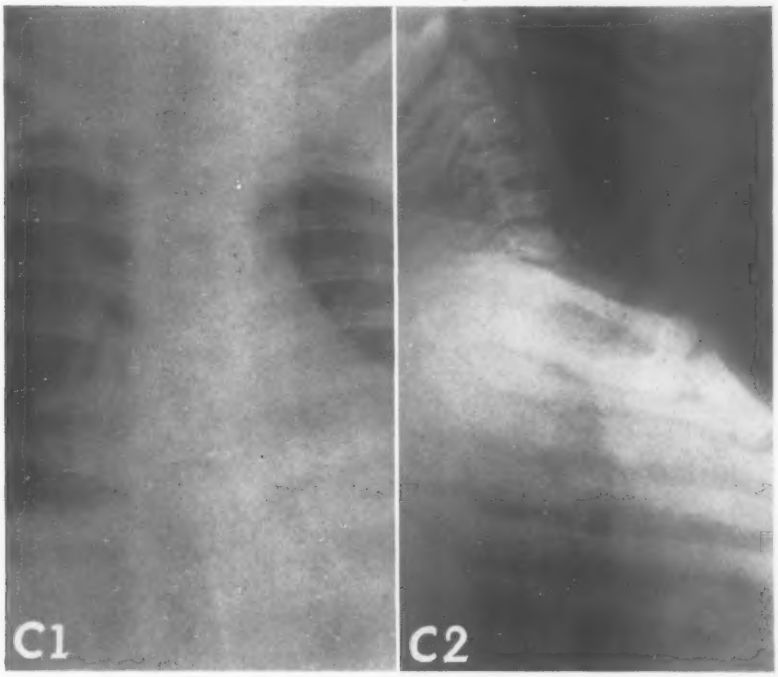

Fig. 7. *A*, large thymus and narrowing of the trachea demonstrated in a six week old infant with noisy breathing, cough and nasal discharge. *B*, similar findings demonstrated four months later. The noisy breathing had persisted in this interval with periods of aggravation during upper respiratory infections. *C*, appearance of the trachea and mediastinum one month after roentgen irradiation of the anterior chest. The noisy breathing had ceased. The thymus is now inconspicuous and the trachea is of large caliber.

CONCLUSIONS

Two cases of congenital tracheal stenosis associated with malformation of the tracheal cartilages have been reported.

The normal infantile trachea usually has a fairly fixed caliber but in some instances transient narrowing of the trachea may occur apparently due to soft tracheal cartilages.

Abnormal cervical and mediastinal masses, a large thymus, or a vascular ring impinging on the trachea will produce stenosis of the trachea depending in large part upon the development and pliability of the tracheal cartilages.*

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Vol. 62, No. 2

CONGENITAL OBSTRUCTIONS OF THE RESPIRATORY TRACT*

II. BRONCHIAL MALFORMATIONS

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IT is reasonable to presume and there is evidence to indicate that atresia and stenosis with malformations of the cartilaginous rings occur in the bronchi as in the trachea. Tracheal malformations have been discussed in a previous communication.[6]

Atresia is much more commonly encountered in a bronchus than in the trachea. It is now a well recognized entity usually designated as an absence, agenesis or aplasia of the corresponding lung. Recent reviews indicate that about 50 well authenticated cases and perhaps over 100 have been reported in the literature.[3,11,25] While the corresponding lung is usually absent it may be present as a hypoplastic remnant. The opposite lung is abnormally large and the mediastinum is displaced to the side of the atresia. This condition is quite compatible with an absence of symptoms and long life. There may be other congenital malformations, however, and malformations of the tracheal cartilages with or without tracheal stenosis have been frequently observed in the reported cases.[3,5,13,14,21,24] The left lung is more often absent than the right in the ratio of about 2 to 1.

Bronchial stenosis as the result of maldevelopment of the bronchial wall has been reported by Overstreet.[17] In his case emphysema was found in the left upper lobe of a three week old infant without symptoms. Dyspnea and cyanosis appeared at one month. The autopsy disclosed emphysema of the left upper lobe with a diffuse marked enlargment of the air sacs and with compression of the left lower lobe and of the right lung. There was a slit-like orifice for the bronchus of the left upper lobe and the cartilage was absent in the posterior wall of the bronchus. Three cases were reported by Ferguson and Neuhauser.[7] In 2 there was obstructive emphysema in the corresponding lung and in 1 there was a sudden death during an operation. In one of these cases an absence of the cartilaginous rings of the bronchus was demonstrated at autopsy and in another "a deficiency of the cartilaginous rings in the locality of the collapsible bronchi" was demonstrated microscopically.

CASE 1. J. C. This infant had a cough and dyspnea since shortly after birth and evidence of a congenital cardiac malformation. There had been repeated admissions to another hospital and there the roentgen examination (Fig. 1*A*) was made at ten weeks of age. The patient was admitted to this hospital at five months of age exhibiting a loud systolic murmur and thrill, a silent left chest, and the roentgen findings shown in Figure 2*B*. Without knowledge of the previous findings the possibilities of agenesis of the left lung or of an atelectasis perhaps produced by the large heart or a vascular malformation were considered. Bronchoscopy showed the walls of the left main bronchus to be collapsed from what was supposed to be an external pressure effect. There was no foreign material in bronchial lumen and there appeared to be no air exchange through the left bronchus. At autopsy two days later a large heart was found with the enlargement involving chiefly the right auricle and right ventricle. The left auricle, which would be the chamber most closely approximating the left main bronchus, appeared normal except for a large interatrial septal defect. There was no malformation of the great vessels and nothing was found to produce a pressure effect on the left main bronchus.

* From the Children's Hospital of Michigan, Detroit, Michigan. Presented in part at the Forty-ninth Annual Meeting, American Roentgen Ray Society, Chicago, Ill., Sept. 14–17, 1948.

Thus it seems proper to assume that there was a deficiency of the cartilage in the bronchial wall to account for the collapse of the bronchus and atelectasis of the left lung. Unfortunately, no particular attempt was made at autopsy to prove or disprove this contention.

Case II. H. A. This infant exhibited a cough at three days of age followed by a wheeze. Both symptoms progressed but it was noted that the respiratory distress was relieved with the baby in an upright position. Respiration was noisy and high pitched. At ten weeks of age laryngoscopy revealed a small web anteriorly in the larynx and this disappeared after passing a bronchoscope.

The respirations were thought to be improved for a week after this procedure but then the wheezing recurred and gradually became worse. At seven months of age the infant was readmitted to the hospital after a spell of severe wheezing and cyanosis. The roentgen study of the chest, which was made only in the posteroanterior projection, showed only emphysema of the lungs and laryngoscopy was not informative. The child died suddenly the following day. At autopsy three cysts were demonstrated at the tracheal bifurcation obstructing both main bronchi (Fig. 2). The cysts seemed to arise from the bronchial walls where there were defects in the cartilaginous rings of both main bronchi. The cysts contained a clear viscid fluid.

Marshall and Cookson[15] reported a tracheobronchial cyst which had presented externally to the tracheobronchial lumen in a female infant with symptoms of cough, cyanosis and dyspnea. The cyst was adherent to the left bronchus and had resulted in atelectasis of the left lung. It had contained a jelly-like material and was lined by a ciliated epithelium. Wallace[23] has reported 2 cases with congenital webs in the bronchi. In one case the web was in the right main bronchus producing atelectasis of the right lung in a three month old female infant with irregular respirations. In the second case the web was in the left main bronchus with atelectasis of the left lung in a one day old female infant exhibiting shallow breathing and cyanosis. Wallace suggested that perforation of the webs and dilatation of the lumen might have been therapeutically effective.

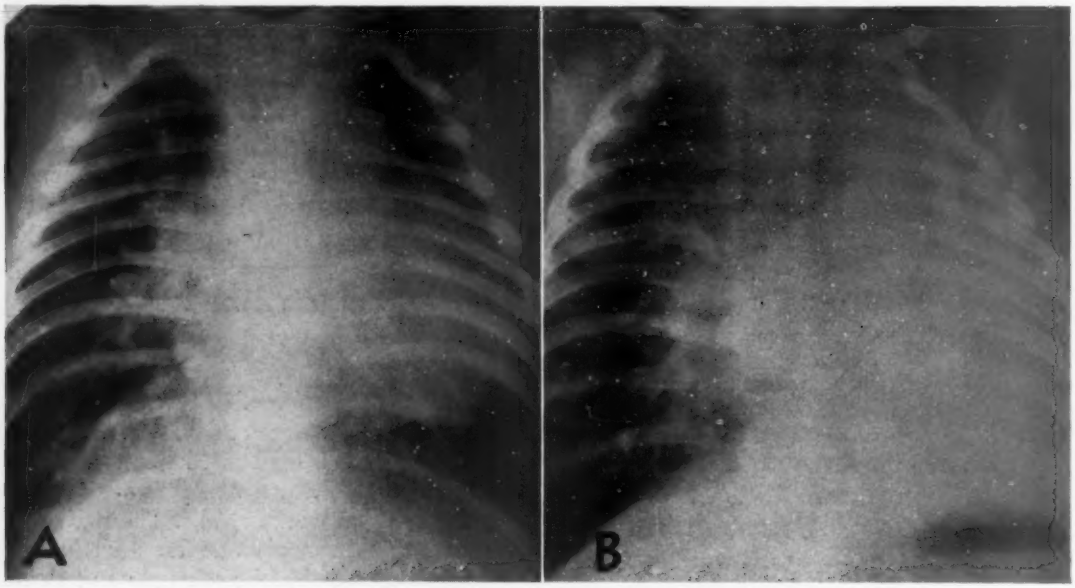

Fig. 1. Case 1. *A*, roentgenogram at six weeks of age showing large heart and congestion in the lungs. *B*. roentgenogram at five months of age two days before death showing massive atelectasis of the left lung, This resulted from collapse of the bronchial wall (bronchoscopy) with no evidence of an intraluminal or extraluminal mass (autopsy) and therefore due presumably to a deficiency of the bronchial cartilage.

The pathogenesis of congenital cystic disease of the lung is unsettled in its details but it seems profitable to consider at least some important forms of cystic disease as a malformation producing stenosis near the termination of one or more bronchial radicles. Formerly the diagnosis of this condition was largely of academic interest but in recent years since it has been demonstrated that surgical intervention is feasible and often successful to the point of saving the life of an otherwise doomed infant the diagnosis has become a more important and gratifying one to make.

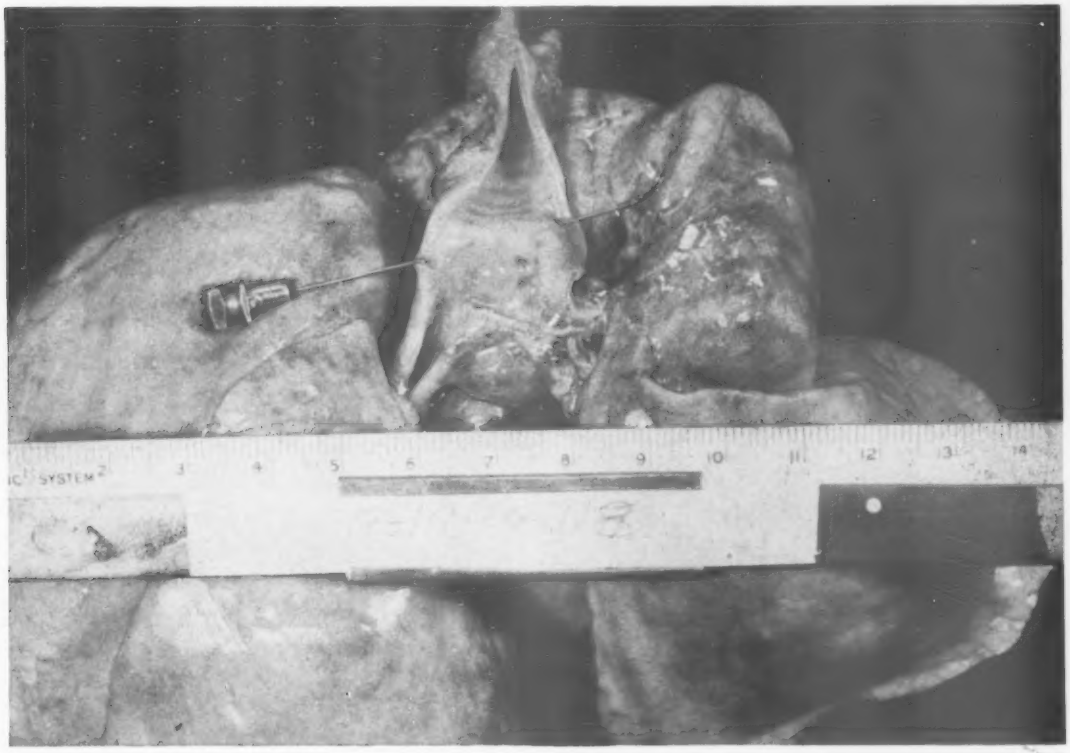

Fig. 2. Case II. Intraluminal bronchial cysts causing obstruction at the tracheal bifurcation. These were associated with defects in the cartilage of the bronchial wall demonstrated at autopsy.

Case III. A. C. This infant experienced a sudden onset of cyanosis and labored respiration at five days of age. On this day the right lung was found to be large and emphysematous with a cystic appearance in the lower half (Fig. 3*A*). It extended across the midline in the upper chest. The mediastinum was displaced to the left and the air content in the left lung was much reduced. Bronchoscopy was not particularly helpful. As the child was observed during the next three weeks there was a progressive if somewhat intermittent aggravation of the respiratory distress, the right lung gradually increased in size and the left lung became apparently completely atelectatic (Fig. 3*B*). Thoracentesis on the right did not aid in the diagnosis or provide relief of the patient's symptoms. It seemed that the right lung was not functioning in spite of its large size and that the only hope for the infant was to sacrifice the right lung and hope that the left lung would resume its function. Accordingly a right pneumonectomy was performed at the age of one month. The child did well following the operation and the left lung re-expanded to a considerable degree (Fig. 3*C*). On the 4th postoperative day the child was comfortable outside the oxygen tent with no cyanosis or dyspnea. Unfortunately the child died on the seventh postoperative day from a tension pneumothorax resulting from a loosened ligature on the right main

bronchus. The autopsy disclosed no other complication or cause of death. The sectioned right lung is illustrated in Figure 3*D*. It is evident that only the larger cysts in the lower lobe had been demonstrated in the roentgenograms. not actually demonstrable in the roentgenograms, that when the cysts are numerous and relatively small the lung may appear simply emphysematous and furthermore that remaining normal pulmonary tissue which has been

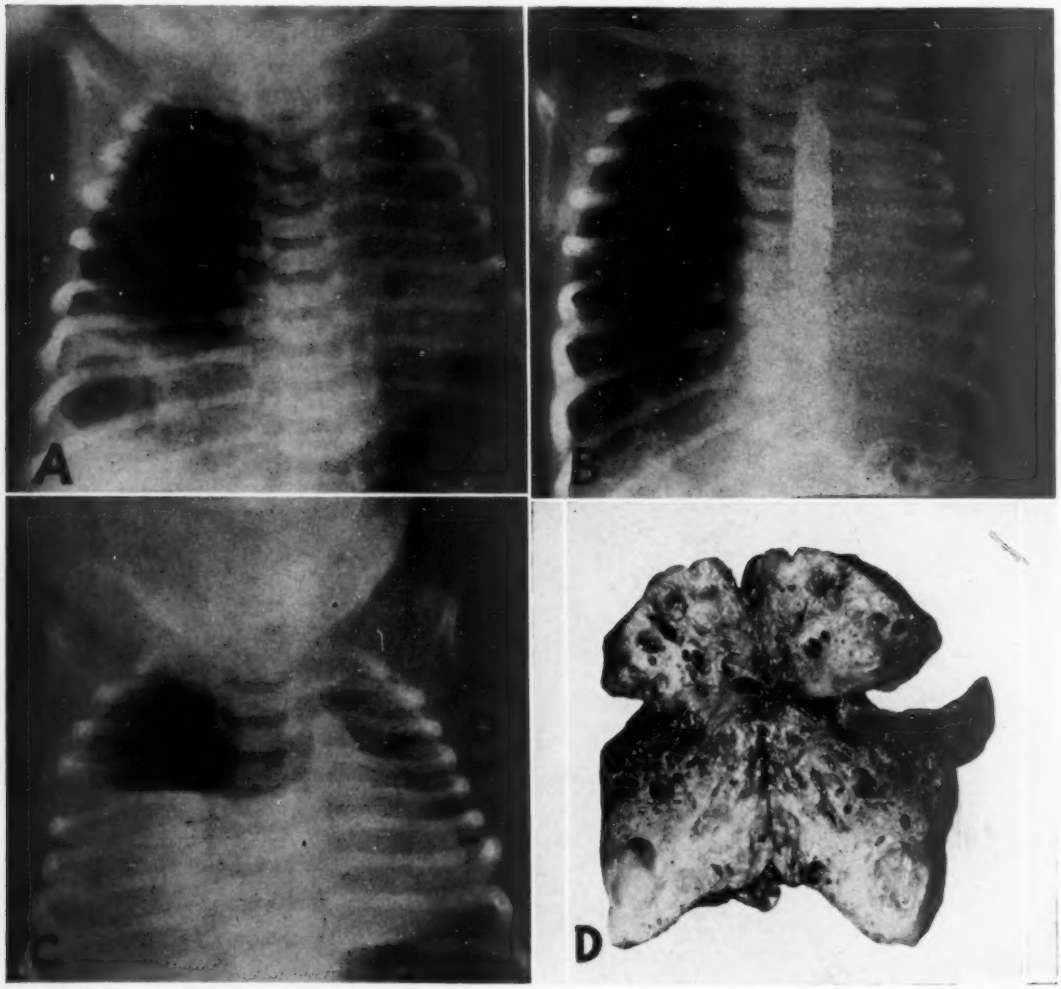

Fig. 3. Case iii. *A*, roentgenogram at five days of age showing large right lung with a cystic appearance in the lower half and displacement of the mediastinum to the left. *B*, examination at thirteen days of age showing increasing size of the right lung, further displacement of the mediastinum to the left and atelectasis of the left lung. *C*, examination at one month of age, two days after a right pneumonectomy showing partial re-expansion of the left lung. *D*, sectioned right lung showing an extensive cystic malformation of the upper and lower lobes and atelectasis of the middle lobe which was otherwise normal.

The upper lobe appeared only as an emphysematous lobe and the collapsed but otherwise normal middle lobe was not evident at all. Microscopically numerous well formed alveoli were observed between the cysts in the cystic lobes. Thus it may be stated that cystic disease of the lung may be present when the cysts are collapsed by the expanding cysts may not be demonstrable.

Case iv. M. W. This infant had labored respirations soon after birth and was thought at another hospital to have an agenesis or atelectasis of the right lung. Certainly the right

lung appeared atelectatic in the roentgenogram (Fig. 4*A*) except for a small basal lateral segment, but we were also impressed by the large emphysematous appearing left lung and it was important to decide whether the symptoms and mediastinal displacement were due to a collapsed or malformed right lung or to a large expanding left lung. No cysts could be demonstrated in the left lung. The symptoms in this case were less severe than those in Case III and at times the baby seemed to be doing well. However, as the patient was observed during the next three and a half months the left lung slowly increased in size and the upper portion of the left lung extended more across the midline into the right upper chest (Fig. 4*B*). Perhaps the anomalous curvilinear shadow at the left base suggests a cystic malformation. With a mild respiratory infection at two months of age there was dyspnea, cough and cyanosis so that the patient was readmitted to the hospital but he was discharged improved after two weeks. Bronchoscopy was not helpful. Finally he was found to be losing weight, there was a persistent cough, and as his existence seemed precarious, a left pneumonectomy was decided upon and was performed at three and a half months of age. He died four hours later. The left lower lobe (Fig. 4*C*) was very large and riddled by multiple cysts from 0.1 to 1.5 cm. in diameter. The left upper lobe was normal except that it had been collapsed to a thin cap over the left lower lobe. It was the impression here that surgical intervention had been too long delayed and that if it had been accomplished sooner the collapsed but otherwise normal portions of the lung would have been more readily able to expand and resume their function.

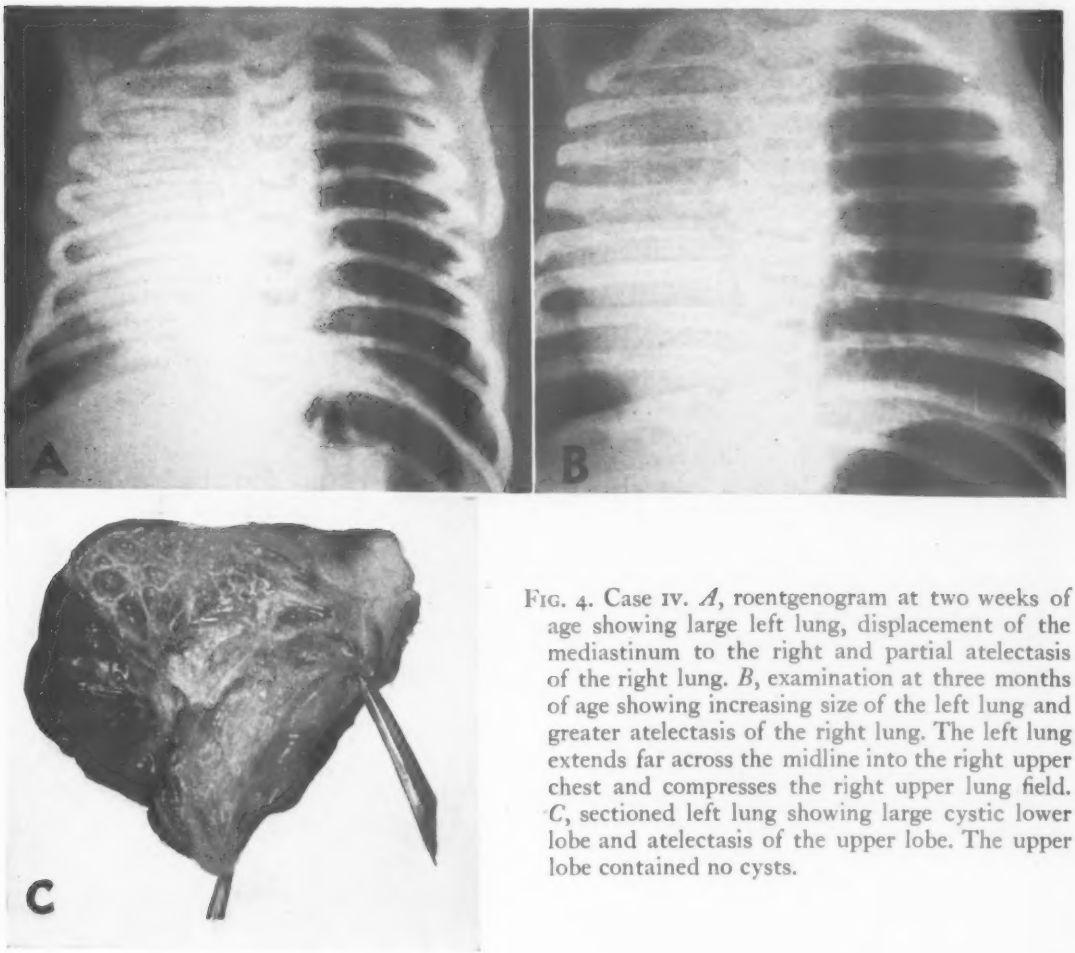

FIG. 4. Case IV. *A*, roentgenogram at two weeks of age showing large left lung, displacement of the mediastinum to the right and partial atelectasis of the right lung. *B*, examination at three months of age showing increasing size of the left lung and greater atelectasis of the right lung. The left lung extends far across the midline into the right upper chest and compresses the right upper lung field. *C*, sectioned left lung showing large cystic lower lobe and atelectasis of the upper lobe. The upper lobe contained no cysts.

Case v. B. N. This infant, a private patient of Dr. John Pollack, was born in July, 1947, and lived through the following winter suffering only mild upper respiratory infections which were in no way alarming. At a routine office visit in April, 1948, Dr. Pollack observed a shift of the mediastinum to the right under the fluoroscope and referred the patient for further study. A large cyst-like cavity in the left upper lobe was demonstrated on the roentgenograms (Fig. 5*A*). It extended across the midline superiorly and displaced the mediastinum to the right. The lower lung field was compressed but there was no evident reaction in the adjacent lung or pleura. Bronchoscopic studies showed nothing remarkable in the tracheobronchial tree. At a subsequent roentgen examination two and a half months later (Fig. 5*B*) the cyst had increased in size, the lungs elsewhere were less well aerated and there was perhaps slightly more displacement of the mediastinum to the right. Operation was deemed advisable to forestall complications of infection and atelectasis, and on July 21, 1948 Dr. Robert E. Gross of Boston excised the cyst in the left upper lobe and closed the defect in the upper lobe by approximating the raw surfaces. To date there has been a satisfactory recovery. This, then, is an example of a single expansile air cyst in the lung which had produced much less severe symptoms, if indeed any symptoms at all, than in Cases iii and iv and one where the diagnosis and the surgical treatment were less difficult.

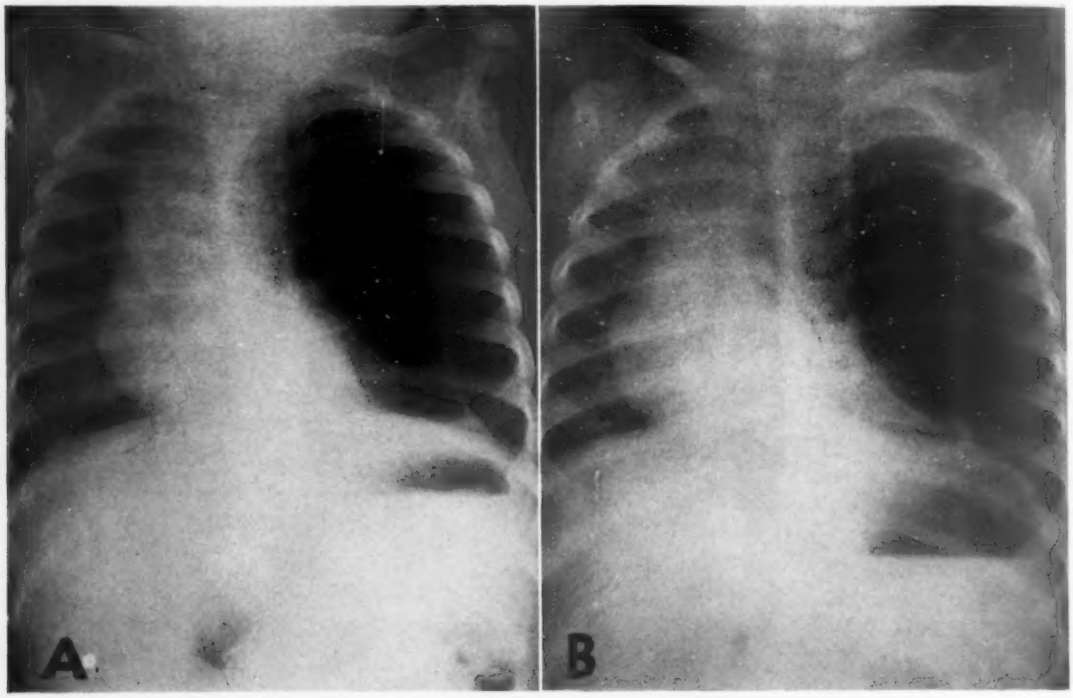

Fig. 5. Case v. *A*, roentgenogram at nine months of age showing large cystic lesion in the upper lobe of the left lung compressing the left basal lung field and displacing the mediastinum to the right. *B*, re-examination at one year of age showing increasing size of the cystic lesion, more displacement of the mediastinum and diminishing aeration of the right lung. A large cyst was successfully removed from the left upper lobe with an uneventful recovery.

In 1926 Miller[16] writing on "Congenital Cystic Lung" stated that "radical cure is out of the question." In 1936 Gale, Keeley and Coon[10] reported a successful pneumonectomy for cystic disease in an adult. In 1943 Fischer, Tropea and Bailey[8] reported a successful lobectomy of the right upper and middle lobes for cystic disease in an infant one month old. In 1946 Gross[12] performed a successful left pneu-

monectomy for cystic disease of the left upper lobe in a three week old infant. Similar cases have since been reported by Burnett and Caswell[4] in Philadelphia and by Brechling[2] in Sweden in 1948.

Surgical removal of the cyst, cystic lobe, or cystic lung now seems the most feasible therapeutic approach for expanding lesions that threaten the function of the remaining lung and also for progressive emphysema without cysts due to bronchial stenosis in such cases as have been reported by Overstreet[17] and Ferguson and Neuhauser.[7] Aspirations may give temporary relief from a single cyst,[1,8,9,16,22] but they are not effective if there are multiple cysts as in Case III. The injection of sclerosing material into the cyst by thoracentesis has not been successful[20] and the attempt to obliterate the bronchial communication of the cyst by the bronchoscopic injection of sclerosing material seems hazardous at best. Cysts may disappear spontaneously,[19] may be dormant without symptoms,[19] or may become infected.[2,18]

In the differential diagnosis of expansile air cysts the following conditions are to be considered: diaphragmatic hernia, pneumothorax, agenesis and atelectasis in the opposite lung, and emphysema due to partial obstruction in one of the larger bronchial radicles. Consideration must also be given to the cavities which are commonly seen in infants and children during the course of pneumonia and which have been variously called acquired cysts, pneumocysts, aseptic lung abscess, lung abscess, localized bullous emphysema, and regional obstructive emphysema. An appearance suggesting congenital cystic disease is sometimes seen in a slowly resolving atelectasis or inflammatory consolidation and in bronchiectasis, except that in these instances the lung tends to be contracted rather than expanded.

CONCLUSIONS

Two cases of bronchial obstruction due to congenital malformations have been reported. In one there was collapse of the bronchial lumen with atelectasis of the lung presumably due to a deficiency of the cartilage in the wall. In another there was partial obstruction due to cysts in the bronchial lumen with defects in the cartilaginous structures of the bronchial wall. Comparison has been made with similar cases reported in the literature.

Three cases of congenital expansile air cysts in the lungs treated surgically have been reported.

Surgical removal of an expanding segment of lung whether due to obstructive emphysema or cystic disease is feasible and desirable when the expanding segment threatens the function of the remaining lung.

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DISCUSSION

Dr. Vincent W. Archer, Charlottesville, Virginia. Dr. Evans has covered the malformations very nicely. There is nothing to be added to this. However, there are two or three things that come up as far as differential diagnoses are concerned which need somewhat more attention.

Dr. Evans did not mention one of the conditions that we always look for since the days of that perfectly beautiful exhibit in Detroit in 1932 by Dr. Pancoast and Dr. Pendergrass on the lateral views of the neck which won a gold medal at the American Medical Association. We have been very much interested in retropharyngeal abscesses. They are not at all uncommon in childhood, and particularly in early childhood as a complication of other infections.

In one of the slides that Dr. Evans showed, it looked to me very much as though there might have been a complicating retropharyngeal abscess. We have found several of these and in the days before chemotherapy they were drained. That was one phase that I think should be considered. Another is what all pediatricians call bronchiolitis in which there is an occlusion of a great part of the smaller divisions of the bronchial tree by mucus, along with infection.

Roentgenologically, you see very little. There is some haziness, generalized haziness of the lung fields, but fluoroscopically you will see quite a marked limitation of the motion of the diaphragms.

Still another infection which, I think, must be considered is diphtheria. We see quite a bit of diphtheria and on the basis of an adult that we saw years ago, we think that we have a fairly definite picture in infections which involve the trachea immediately below the larynx.

In this patient, it was a question of malignancy and she was biopsied and it was inflammatory, and on the roentgenogram we saw this dumb-bell shaped constriction of the trachea. We treated her with radiation similar to that described by Dr. Osmond for thyroiditis and we had a very prompt recovery. During the last year, there was an epidemic in one locality. Several of the children brought over to us came with the thought that they had diphtheria. Cultures were negative and yet all of them showed the same dumb-bell narrowing of the trachea but immediately beneath the larnyx. Those children, we believe, had an infection in the wall of that trachea analogous to the one that I described which was proved bacteriologically.

There is one thing which might be mentioned. All of us see these children when they come in, with noisy respiration, etc. I think it is important to look at them during the phases of respiration and if you see a child who is struggling for breath, and has a dip-in of the xiphoid on respiration, then it is necessary to carry out all of these things in order not to miss the diagnosis since there may be a foreign body or some stricture either involving a large portion of the bronchial tree or above the bifurcation of the trachea.

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LESIONS OF THE DIAPHRAGM*

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THE diaphragm is a musculofibrous dome-shaped structure which is attached to the lower margin of the thorax and to the upper lumbar vertebrae, arising embryologically in the region of the neck, and maintains cervical relations through its innervation by the phrenic nerves, which spring one on each side, usually from the third to the fifth cervical nerves. The central portion of the diaphragm is an aponeurosis or central tendon into which is inserted the musculature which arises from the ziphoid cartilage, the cartilages and tips of the last six or seven ribs and by means of the crura from the sides of the first four lumbar vertebrae on each side. The diaphragm presents three main openings. Anteriorly and to the right is the opening for the inferior vena cava, posterior to the opening and in the midline is the esophageal opening. The aortic opening is posterior to the esophageal opening and is actually behind the diaphragm. Anteriorly to each side of the sternum the foramen of Morgagni is located. The central portion of the diaphragm is frequently absent; occasionally the diaphragm is incompletely developed dorsally on the left side. Defects of the right diaphragm are not common.

During the last decade, diaphragmatic hernia has become one of the common gastrointestinal conditions encountered in hospital practice. Prior to the extensive use of the roentgen ray, hernias and other lesions of the diaphragm were not commonly suspected and were identified only at autopsy. As the importance and accuracy of roentgen diagnosis was appreciated, the internist became aware of the symptoms often accompanying diaphragmatic hernia, and at the present time many cases are accurately diagnosed when sent to the roentgenologist.

The symptoms caused by hernia are varied and bizarre, and the size of the hernia has much to do with the severity of the symptoms. The early diagnosis of a small hernia is very important because of the possibility of the hernia becoming larger if proper treatment is not instituted. If a patient with a small hernia is allowed to take large quantities of food and fluids and then lies down, the intragastric pressure increases and the hernia becomes larger. I am sure you are all familiar with the cardiac symptoms caused by or at least accompanying diaphragmatic hernia. According to our cardiologists, symptoms develop which are often difficult to differentiate from angina or the pain from coronary thrombosis. The symptoms are often transient probably due to the fact that the hernia is of the recurrent or intermittent type; in other words, the hernia may be present at one time and absent at others, the "sliding type." The reason that the hernia can be found by one roentgenologist and not by another, even though both examinations were very thorough, is that the stomach may not herniate at all times. In some patients, however, the hernia may be fixed due to adhesions.

Diaphragmatic hernias are usually found after the fourth decade of life about equally divided between males and females. Rigler has found a high incidence (18 per cent) in 195 women during pregnancy. Diaphragmatic hernias are very infrequent before forty years of age. Those seen in children are usually due to large developmental defects through the dome of the diaphragm and are not common through the esophageal opening. Diaphragmatic lesions we have seen in children have been due to absence of all or part of the diaphragm. Most of the patients were seen

* Carman Lecture, presented before the St. Louis Medical Society, St. Louis, Mo., November 4, 1947.

when they were very young and the lesions were undoubtedly of the congenital type. LeWald has reported a marathon runner with an absence of the diaphragm on one side.

Diaphragmatic hernias are either congenital, acquired or traumatic. We believe that those which develop later in life are acquired, whereas the congenital type occurs in infants and the symptoms are very pronounced and death often occurs, especially if the opening is very large and most of the abdominal viscera are in the chest. If the diagnosis is made promptly, our present day thoracic surgeons can operate and repair the rent in the diaphragm in the very young infant.

The traumatic hernia is due to an injury and does not commonly involve the esophageal opening. The diagnosis is not difficult, and this type of case is met with more in traumatic surgery than in general medicine. The hernia, or better, evisceration, does not as a rule involve any of the normal openings of the diaphragm; usually the dome is torn. Herniation of the viscus usually occurs through one of the foramina of the diaphragm. The esophageal and Morgagni are the usual sites, although the esophageal is by far the commonest location.

Congenital hernia usually occurs through the left diaphragm as the liver protects the right diaphragm, and even though a defect may be present, the liver blocks the entrance of a viscus into the thorax. We have seen only 2 cases of right-sided hernia, one was in a stillborn with an absence of the right diaphragm and the other in a woman who had assumed a knee-chest position for many months allowing the transverse colon to herniate through an opening in the right diaphragm.

Hernias through the esophageal opening of the diaphragm are now thought to be of the acquired type. They have a sac composed of one or more of the layers of the diaphragm. Hernias occurring through the foramen of Morgagni, "para-sternal," are also classed as acquired and usually have a sac. Congenital hernias due to large developmental defects are devoid of a sac and have the serous coat of the viscus as a cover. The traumatic hernia or evisceration likewise has no sac.

Many factors have been attributed as the cause of acquired diaphragmatic hernia. I will mention some of the more common as taken from the literature. Increased intra-abdominal pressure is given as a common cause by many writers. Decreased intrathoracic pressure has also been given as a cause. The diaphragmatic esophageal membrane at the esophageal opening is quite strong in early life and this may account for the infrequency of diaphragmatic hernia during childhood. Later in life the membrane becomes thinned and with increased intra-abdominal pressure the stomach is forced upward into the chest. With relaxation of the tissues about the hiatal orifice, one can see why an esophageal hiatal hernia may occur with increased intra-abdominal pressure due to obesity, especially in a patient with a muscular abdominal wall.

Gilbert and his associates found it difficult in the "experimental animal" to produce a hernia with the chest closed and the abdomen open. They doubt the importance of decreased intrathoracic pressure as a cause *per se*. Deep inspiration in constant cough might be a factor as well as the increased intra-abdominal pressure. Difference in pressure between the thorax and abdomen may be a contributing factor. Von Bergmann some years ago suggested that many hernias might be due to traction on the esophagus from above in response to vago-vagus reflex initiated by some stimulus originating in the upper abdominal viscera.

The longitudinal fibers of the lower esophagus are continuous with the longitudinal fibers of the stomach and are motivated by the vagus nerve shortening of these fibers which would tend to pull the stomach upward and through the relaxed hiatal orifice.

Kuckuck quoted by von Bergmann stimulated the vagus trunk in the neck of the rabbit and was able to demonstrate a shortening of the esophagus, and in some

cases the cardiac end of the stomach extended above the diaphragm. The esophagus is very susceptible to nervous stimuli as witness globus hystericus, cardiospasm and esophageal symptoms occurring reflexly in early stages of carcinoma of the stomach.

Gilbert and his co-workers confirmed the work of Kuckuck in dogs. The vagus trunk in the neck was electrically stimulated, the esophagus was shortened and the stomach was pulled up to but not through the hiatal opening. The chest was closed in the experimentation. Later when the chest was opened, the stomach was pulled up through the hiatal orifice. Later in some further work, Gilbert and his co-workers produced shortening and lengthening of the esophagus recorded on a drum by a lever fastened to the esophagus.

Irritation of the peritoneum by scratching, manipulation of the lobes of the liver, distention of the gallbladder and stretching of the stomach wall all caused a shortening of the esophagus and a pulling up of the stomach to and in some cases through the hiatal orifice. Other writers have attributed many conditions as the cause of hiatal hernia such as fatigue, nervous factors and vagotonia. Personally, we believe the diaphragmatic membrane which relaxes with age accompanied by obesity are two very important causes.

In Gilbert's series of 48 cases, all carefully studied, 27 in which there was some possible source of vago-vagal reflex, in 2 of the cases the hernia was incarcerated, in 3 patients there was evidence of blood in the stools. Thirty of the patients came under observation because of gastrointestinal symptoms, 16 came under observation because of the symptoms of angina pectoris and 2 because of paroxysmal auricular fibrillation which came on when the hernia was present. Stooping over is frequently a cause of the symptoms, especially in the obese. Reduction in weight often caused relief of symptoms.

ROENTGEN DIAGNOSIS

Careful roentgenologic observation is imperative if hiatal hernias are to be identified. Hernias are often recurrent, and it may be necessary to examine a patient many times before the hernia is found. Even the fixed type of hernia can change.

The identification of the stomach folds is the deciding factor in the differentiation of the hernial sac from a large phrenic ampulla; this is not always easy, especially in large patients.

The roentgenoscopic examination of the diaphragm reveals normal excursions in the presence of hiatal hernia. In the congenital type with a large rent, the excursions are often impaired. Hernias through the central tendon of the diaphragm often cause changes in the excursions.

Frequently the possibility of a hernia is suspected during the initial roentgenoscopic examination before the barium meal is given. Gas can be seen above the diaphragm adjacent to the cardiac opening. This is not a constant finding if the hernia is of the recurrent type.

The esophagus, in our experience, is usually long and redundant in hiatal hernias. If the hernia is of the peri-esophageal type, the stomach passes upward around the esophagus, displacing the esophagus to the right and backward, entering the stomach below the diaphragm. If the hernia is of the direct type, the esophagus is carried upward and is usually redundant in the chest and enters the stomach above the diaphragm. This type is often confused with a congenital short esophagus. In congenital short esophagus, the herniated portion of the stomach is always above the diaphragm; in other words, it is developed in the chest and has failed in its downward transit. It can be classed as a partial thoracic stomach.

The differentiation of hernia and congenital short esophagus is important from the surgical standpoint. If the esophagus is of the congenital short type, it will be very difficult, if not impossible, to bring the stomach down into the abdominal cavity. The surgeon should have the information before considering surgery—a phrenic exeresis will be more helpful in the congenital short esophagus.

The phrenic ampulla has been incorrectly diagnosed congenital short esophagus, and at times it may be difficult to differentiate the two conditions due to the band-like constriction at its upper margin, not unlike esophageal sphincter. The phrenic ampulla is a functional or physiological bulbous-like dilatation of the lowermost portion of the esophagus. It is demonstrated where the esophagus is completely or moderately well filled. It is apparently not responsible for clinical symptoms and is of importance only in that it must be recognized as such and not confused with a hernia or a congenital short esophagus which it may closely simulate. The rugae of the esophagus are quite characteristic and are quite readily demonstrated with almost any type of barium meal. They appear as continuous, long, thin lines of barium and are not often branched or broken in appearance. Phrenic ampulla is common after forty years of age in a hypersthenic person. It is exaggerated by rapid swallowing of the barium or by increased intra-abdominal pressure, such as performing the Valsalva experiment. The ampulla partially or completely empties at the end of the act of swallowing. The most important roentgenological finding in the differentiation of phrenic ampulla and congenital short esophagus and hernia is the rugal pattern seen either roentgenoscopically or roentgenographically. If the rugae are parallel with the long axis and not broken, one can feel sure he is dealing with a phrenic ampulla and not a part of the stomach.

Thoracic stomach is a developmental defect in which the entire stomach is permanently in the thorax. The esophagus is very short and the pyloric portion of the stomach passes through the esophageal opening of the diaphragm. The condition is compatible with life. One of the patients we have in our series was sixty-seven years of age at the time of examination. All he complained of was fullness in the chest and a desire to expectorate due to the fact that the fluid came up into his mouth.

To identify these diaphragmatic lesions it is necessary to be very meticulous, in other words, be hernia conscious. While some cases can be readily diagnosed in the upright position, a large percentage will be missed if patients are only examined in the upright position. Every patient in whom a hernia is suspected must be roentgenoscoped and roentgenographed in the recumbent position, including both anteroposterior and posteroanterior views in the Trendelenburg position. During roentgenoscopy, the patient should be asked to strain, bear down as when going to stool. Also, the Valsalva experiment is very useful. The right lateral and the George position is also of great value during roentgenoscopy. The length of the esophagus and its relation to the hernia can be determined in these positions.

SUMMARY

Diaphragmatic hernias are not uncommon and may be accompanied by very bizarre symptoms often simulating angina pectoris and coronary disease. The identification of hernias is fundamentally a roentgenological problem, and frequent careful examinations are often necessary before the hernia can be diagnosed. Hernias are very often of the recurrent type. Congenital hernias are usually devoid of a sac, whereas the acquired type usually has a sac.

Seventy-eight per cent of non-traumatic hernias occur on the left side. Twenty-five per cent are through the esophageal hiatus, 25 per cent occur posteriorly and about one-sixth are central. Twenty-two per cent of hernias occur on the right side and 25 per cent are at the central position, 25 per cent posterior and one-third at the parasternal foramen. Ninety-five per cent of traumatic hernias are on the left side, and nearly 50 per cent of them are lateral and 25 per cent central. About 50 per cent of the traumatic hernias that occur on the right side are situated centrally. Fifty per cent of bilateral hernias are through the parasternal openings.

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RETICULO-ENDOTHELIOSIS

HAND-SCHÜLLER-CHRISTIAN DISEASE AND THE RARER MANIFESTATIONS

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RETICULO-ENDOTHELIOSIS, or hyperplasia of the cells of the reticulo-endothelial system, is an entity occurring sufficiently frequently in general hospital practice to warrant the interest of every clinician. This interest should be increased by the fact that, in many cases, the diagnosis is completely missed during relatively long periods of hospital observation. Further, there are numerous instances in which pathological examination of biopsy or necropsy material is so inconclusive as to be of no actual value. Except in the most clear cut involvement of a characteristic group of organs, as occurs in the Hand-Schüller-Christian variation of this disease, diagnosis is usually only made by the alert roentgenologist and all too frequently his findings are disagreed with by both his clinical colleagues and the pathologists who, however, are never able to substantiate a more tenable diagnosis.

On the roentgenologist's part, diagnosis is made by recognition of entirely typical defects, usually in the membranous bones (Fig. 1), though the long bones may also be involved. The soft tissue changes in reticulo-endotheliosis are far more widespread than those in bone, but unfortunately are not readily demonstrable by roentgen examination as are the bone changes.

Reticulo-endotheliosis is of even greater interest to the roentgenologist because of the fact that the only known satisfactory method of treatment lies in the field of radiation therapy, with quite constant palliation and improvement. There is no other group of diseases in which the roentgenologist is so intimately and essentially concerned with both diagnosis and treatment, though the first description of autopsy findings in a variant member of the group was made by Hand[4] in 1893 two years before Röntgen's announcement of his discovery.

Hand's original case was complicated by a generalized acute tuberculous infection and was thought to be a manifestation of unusual reaction to the tubercle bacillus, though characteristic lesions were described in the autopsy material, and exophthalmos, diabetes insipidus, and a defect in the right parietal bone were demonstrated in the living patient.

Schüller[11] in 1915 described a patient

FIG. 1.

presenting unilateral exophthalmos, bony defects in the calvarium, and typical dystrophia adiposogenitalis; also a patient showing exophthalmos, cranial and pelvic bony defects with a healing pathological fracture of the femur, and marked diabetes insipidus. His conclusion was "Anomaly of the skeleton as a result of disease of the hypophysis."

In 1918, Christian[2] described a patient with a two year progressive history beginning with loosening and decay of the teeth with tender swollen gums, with subsequent exophthalmos, diabetes insipidus,

headache, and gradual loss of hearing, with typical roentgen changes in the cranial and pelvic bones. On comparison of his case with the 2 cases reported by Schüller, Christian concluded that his patient was entirely similar to those of Schüller, and that the findings could be explained wholly on the basis of pituitary dysfunction. For a number of years after Christian's publication, scattered additional cases were described and the hypothesis of a pituitary etiology was widely held.

Rowland,[9] in 1928, correlated 2 cases of his own with the 12 already reported, and with other cases of lipoid reticulo-endotheliosis and concluded that all were variations of the same clinicopathological entity. This conclusion was reached on the basis of lipid deposit, in all the members of the group, in areas abounding in reticulo-endothelial cells. In individual cases, the extent of deposit in any single location showed a wide variation.

For many years, the curiosity of the histopathologists has been piqued by the presence, the origin, and the function of certain heteromorphic phagocytic cells which lie loosely in the crevices of tissues where they may wander leisurely or whence they may enter the circulating blood stream. These have been variously described[7] as wandering cells (clasmatocytes), adventitial cells, macrophages, histiocytes, endothelial leukocytes and polyblasts. Recognizing the abundance of these cells in certain specific somatic locations, Aschoff assumed that there is a definite reticulo-endothelial system, with localized collections of these phagocytes, which, as members of the system, he termed reticulo-endothelial cells. Their presence in large numbers in specific locations and their active phagocytic properties are readily proved experimentally by injection into the blood stream of any undissolved material. The largest collections of the reticulo-endothelial cells occur in the sinuses of the spleen, the bone marrow, the lymph nodes, and the lungs. The Küpffer cells of the liver are morphologically and functionally indistinguishable from the free wandering members of the group.

Maximow and Lewis[6] have observed, in vivo and in cultures, the growth and development of these cells from a single small mononuclear structure resembling a lymphocyte to giant multinucleated protoplasmic masses, by the relatively simple processes of splitting and fusing of several, combined with engulfing and ingestion of foreign material.

Although the process of fat metabolism is incompletely understood, it is recognized that many ingested fats pass through the intestinal walls unchanged and enter the blood and lymph streams as undissolved foreign material. As such, they are engulfed by the phagocytes of the reticulo-endothelial system and are found in abundance in the reticulo-endothelial cells. This has been experimentally proved by feeding large amounts of cholesterin to experimental animals. Aschoff assumes that the cells themselves control the deposit of fat within them, and therefore exert a very great influence on intermediary fat metabolism.

Rowland[9] hypothesized the development of so-called Hand-Schüller-Christian disease in this way: with a hyperabundance of undissolved foreign material in the circulating blood (in this case cholesterol lipids), there is injury of vascular linings with perivascular infiltration with large numbers of phagocytic reticulo-endothelial cells, attempting to remove the irritant material from the circulating blood. The involved areas grow rapidly because of coalescence of adjacent cells and ingestion of smaller or damaged cells by the larger ones. This soon produces capillary delay with subsequent death of additional cells and their consolidation into the phagocytes. New phagocytes constantly arrive in the area via the blood stream, giving rise to the characteristic microscopic arrangement of smaller, newer cells in the center of the lesion near the vessels, with larger cells more full of lipoid material in the periphery. The larger cells, being displaced from their blood supply, frequently undergo aseptic necrosis and are replaced by fibrous tissue, which probably accounts for the microscopic picture of diffuse fibrosis frequently seen

in the more chronic instances of so-called Hand-Schüller-Christian disease.

It is very interesting, at this point, to conjecture whether the improvement seen after radiation therapy in these conditions is not due to intimal damage by irradiation to the capillaries of the region, with a gross diminution in blood supply to the lesions, hastening the process of aseptic necrosis and fibrous tissue replacement.

Wallgren[15] disagreed thoroughly with Rowland's theory of a hypercholesteremia as the primary phenomenon in Hand-Schüller-Christian disease and cites numerous opinions that lipoid deposit in the phagocytic cells is a secondary and local phenomenon, and that idiopathic abnormal infiltration of reticulo-endothelial cells is the primary lesion in the disease, with secondary ingestion of blood cholesterol to form typical foam cells in certain organs. As proof, Wallgren points out the characteristic occurrence, in the same patient, of areas of marked reticulo-endothelial proliferation with large numbers of cholesterol-containing foam cells in certain organs, while other organs in the same patient show marked reticulo-endothelial proliferation with complete absence of foam cells. Similarly, hypercholesteremia is not seen in every case of Hand-Schüller-Christian disease and has been described in other cases of reticulo-endotheliosis in which there is no lipoid storage. Both of these facts seem to indicate that hypercholesteremia, when present, results secondarily from proliferation of reticulo-endothelial cells. One of the cases to be cited later by the author showed a terminal hypercholesteremia after a more or less typical chronic non-lipoid reticulo-endotheliosis became suddenly acute, with fulminating liver involvement.

Wallgren further disagrees with the time-honored hypothesis that classifies Hand-Schüller-Christian disease as a xanthomatosis belonging to the group of lipoid-storage diseases, including Gaucher's and Niemann-Pick's disease, and xanthelasma, chiefly because of the fact that, in the true xanthomatoses, a quantitative increase in the stored material is always demonstrable in the blood and non-involved tissues, whereas hypercholesteremia is definitely an inconstant finding in Hand-Schüller-Christian disease. His conclusions are substantiated by very extensive investigation by a number of different workers.

Abt and Denenholz[1] have reviewed a number of cases of an obscure acute disease occurring in infants and young children, usually rapidly fatal, characterized by generalized reticulo-endothelial hyperplasia. They found, in some cases, an acute infection which could be regarded as the factor responsible for the great increase in cells of the reticulo-endothelial system. In other cases, there was no evidence of an etiologically significant infectious process. This latter group of cases, they identified as Letterer-Siwe disease, in honor of its earliest investigators.

Letterer-Siwe disease is characterized, according to Siwe[12] by: (1) splenomegaly and hepatomegaly; (2) hemorrhagic diathesis; (3) generalized non-infectious lymphadenopathy; (4) progressive secondary anemia without leukocytosis; (5) occurrence in infants and young children, fatal prognosis, and variable duration; (6) localized skeletal changes, and (7) generalized reticulo-endothelial hyperplasia without constant storage of lipoid material. Wallgren denies the existence of a clinical entity of infectious non-lipoid reticulo-endotheliosis, claims that in all reported cases any associated infection is merely concurrent, and that all the evidence is to the effect that the so-called infectious and non-infectious (or Letterer-Siwe) types are identical.

Wallgren further points out that the stage of development in either Schüller-Christian or Letterer-Siwe disease largely determines the question of cholesterol deposit in the hyperplastic reticulo-endothelial cells. He interprets the well known facts of foam cell formation in some organs while others are not affected, and the change in the histopathologic picture in individual cases during the course of a protracted disease, when non-lipoid reticulo-endothelial hyperplasia may be present at biopsy with typical foam cell

formation at necropsy, and when areas showing typical foam cells at biopsy subsequently show fibrous tissue scarring, as indicating that there is a single disease, namely reticulo-endotheliosis. If it starts in infants or very young children, the typical fulminating course of Letterer-Siwe disease is the characteristic sequence of events, and, in older individuals, the characteristic protracted course is seen, with much less prominent symptoms, and possibly secondary cholesterol deposit in foam cells in any involved organ.

The pathological findings vary only in the presence or absence of stored cholesterol —the types of cells which become hyperplastic are identical in the two conditions, the locations of the deposits of hyperplastic reticulo-endothelial cells are likewise identical, and the resulting symptoms and findings are so much alike that the two diseases in varying forms cannot always be distinguished. Though Letterer-Siwe disease, in every reported case, has run a rapidly fatal course, there are many cases of Hand-Schüller-Christian disease running a subacute course rather than the typically described chronic course and there are cases in the literature varying all the way between the two.

There is so much clinical, pathological, and reasonable evidence in favor of Wallgren's stand that it seems highly probable to the author that there is one pathological condition of reticulo-endothelial hyperplasia, its findings modified by age of the individual, organ or area of deposit of hyperplastic cells, and by some other factor of repair, as obscure as the factor causing original reticulo-endothelial hyperplasia.

It seems highly logical to suggest that cholesterol deposit, with production of foam cells which become immobilized reservoirs, is a reparative attempt on the part of the organism as a whole to counteract the unbridled production of reticulo-endothelial cells. The immobilized reservoirs frequently are located in areas not too significant or important to the physiology of the individual as a whole. Along this line, in Letterer-Siwe disease, the power to immobilize these hyperplastic cells by lipoid deposit within them, would seem to be lacking in the very young. Conversely, as would seem the case in 2 of my patients, after a protracted course, for some unknown reason, probably fatigue, this reparative ability is lost, and generalized involvement and infiltration of vital systems follows rapidly, with inevitable results. Similarly, in cases of Hand-Schüller-Christian disease which are not fatal, this reparative process of cholesterol storage and immobilization of reticulo-endothelial cells may continue as long as the reticulo-endothelial system is stimulated to hyperactivity—once the hyperplasia stops, the cholesterol is absorbed and the deflated cells are replaced by fibrous tissue.

Gross and Jacox[3] have reported at length on the striking anatomical, histopathological, symptomatic and roentgenological similarity between so-called eosinophilic granuloma and the described types of reticulo-endotheliosis, and have concluded that eosinophilic granuloma is another reticulo-endotheliosis with a tendency to predominant hyperplasia of the eosinophilic elements, and a usually solitary bone lesion, though much soft tissue involvement may be present and undiscovered. No autopsies have been reported on patients with eosinophilic granuloma.

It is of particular interest that one of my cases, with multiple bone lesions, had a biopsy diagnosis of eosinophilic granuloma. This patient's history extended over almost three years, and there were associated findings of a moderate anemia, generalized lymphadenopathy, and malnutrition and delayed development.

The experimental work along the lines of reticulo-endotheliosis is meager but most suggestive in its implications. Rowland[10] quotes the artificial production from reticulo-endothelial cells of typical xanthoma cells, in rabbits, by Anitschkow and Chalatow, following high cholesterol diets. Sosman[13] quotes further independent work by Yuasa, feeding cholesterol to higher

animals, with the same results. Much more recently, Muyaji[8] has shown, with rabbits, that hypercholesteremia alone will not cause lesions comparable to lipoid granulomas. Artificially produced inflammatory granulomas show a tendency to small deposits of cholesterol, which are incomparably greater in the presence of hypercholesteremia.

Sosman,[13,14] Kennedy,[5] and Gross and Jacox,[3] in subsequent studies of their own and many other patients, have pointed out that many symptoms and findings are compatible with the diagnosis of Hand-Schüller-Christian disease; that not all are found uniformly; and that not even Christian's triad of skull defects, exophthalmos, and diabetes insipidus is found in every case. This is readily explained by the concept of chance deposit of large numbers of reticulo-endothelial cells in various locations.

I have been very fortunate in seeing 7 patients showing more or less typical skull defects. The only other common findings in all the cases were slight eosinophilia and moderate anemia. There was no apparent influence of age or sex, but 2 patients with rather typical Hand-Schüller-Christian disease were Mexican, and the single Negro patient showed skin lesions. None of the patients showed any marked hypercholesteremia at first admission, though one who died an acute death with liver involvement showed a very high cholesterol value terminally. The majority of the patients showed defects in bones other than the skull, especially mandible and maxilla, diabetes insipidus of varying degree, gingivitis, growth delay, and malnutrition. The less frequently occurring findings included exophthalmos, jaundice, aural discharge, generalized lymphadenopathy, hemorrhagic diathesis, skin lesions, and a variety of intercurrent conditions. Hepatosplenomegaly was not a prominent feature.

None of my cases came to autopsy, but it is extremely interesting that, in the 5 cases where satisfactory biopsy was obtained, there were two positive pathological diagnoses of Hand-Schüller-Christian disease, one of eosinophilic granuloma, and two perfect descriptions of reticulo-endotheliosis without characteristic lipoid deposit and foam cell formation.

All but one of the cases have received roentgen therapy under my direction, with varying results.

CASE REPORTS

Case 1. F.C.B. This white male, aged twelve, was admitted to John Sealy Hospital, Galveston, Texas, on December 19, 1942, with a chief complaint of a painful swelling behind the right ear, present for four months. Additional complaints were discharge from the right ear for two months, and loose lower teeth, also for two months.

The parents stated that this was their twelfth child, that he had suffered the usual childhood diseases, and that his development and growth had been perfectly normal for his first eight years. At the age of eight, they began to notice polydipsia and polyuria, and failure to grow and gain weight. At that time, there was a non-tender swelling on the vertex, which subsided spontaneously. During the past four months before admission, he had lost 20 pounds.

Physical examination showed: (1) a hot tender swelling over the right mastoid region; (2) loose dirty teeth with gums draining purulent material; (3) palpable defects in the right parietal bone; (4) draining right ear; (5) emaciation; (6) stomatitis with a raw, cracked tongue.

Laboratory findings were: (1) urine, specific gravity of 1.002 to 1.006; (2) leukocytosis of 14,000 with 3 eosinophils per field; (3) anemia of 3,800,000, with 73 per cent hemoglobin; (4) *Necator americanus* in the stool; (5) slight elevation of the glucose tolerance test.

On admission, sulfanilamide was started by mouth, and a total of 9.5 grams was given, without significant change. On December 21, 1942, a complete right mastoidectomy failed to reveal any infected material, and the mastoid cells were seen completely replaced by a reddish-gray tumor mass. Tissue specimens unfortunately were autolyzed by the time they were examined. After two postoperative blood transfusions, irradiation of the skull was started on December 24, 1942.

Roentgenograms of the skull showed, on admission, a large irregular punched-out defect in the right mastoid region, and multiple similar

Fig. 2. Case 1. Roentgenogram of the skull. Note parietal and occipital defects.

defects in the right parietal bone (Fig. 2). Roentgenograms of the right mandible showed marked excavation about all the roots of the teeth (Fig. 3). In none of the lesions was there any evidence of any new bone formation. The diagnosis given was "Schüller-Christian disease." Between December 24, 1942, and January 4, 1943, a total of 1,000 r, measured in air, was given to each of two 10 by 10 cm. fields, in the temporoparietal regions, at 217 kv. (peak), constant potential, 18 ma., 50 cm. skin target distance, using filtration of 1 mm. copper and 0.5 mm. aluminum, with a half-value layer of 1.5 mm. copper.

The patient remained in the hospital, showing daily improvement in all his findings, except the diabetes insipidus, with daily intake and output of about 8,000 cc.

A secondary closure of the right mastoidectomy was done on February 2, 1943.

On February 25, 1943, there was a generalized exacerbation of all the symptoms, with several new areas appearing in the left side of the skull, increase in intake and output, pain in the jaws, and pain in the right mastoid region.

Between March 2 and March 6, the patient was given another 750 r to each of the two temporoparietal fields, including the mandible, using the factors described above.

Following this additional therapy, there was marked improvement, with regression of all the symptoms, and the patient left the hospital on March 27, 1943.

Between March 27 and May 6, patient remained at home with decreasing polydipsia and polyuria and few other symptoms except difficulty in eating due to the sore, loose teeth. Diplopia was experienced on one occasion, and there was a complete temporary alopecia.

The patient was re-admitted on May 6 with another acute exacerbation. There were no significant differences, on physical examination, from the findings on previous admission. Several loose teeth were removed.

Between May 12 and May 17, a total of 500 r was given to each of the lateral skull fields, using the same technique as described above, and patient was discharged slightly improved on May 22, 1943.

Roentgenograms made just before discharge showed no change in the appearance of the cranial and mandibular lesions. There were two admissions, from June 20 to June 30, and July 4 to July 5, for treatment of stomatitis and removal of additional teeth. Intake and output had dropped to about 2,000 cc. and no additional skull lesions had appeared. The patient has not been seen since July 5, 1943.

Comment. Most striking in this patient were the absence of exophthalmos, the marked stomatitis most probably due to a

Fig. 3. Case 1. Right mandible.

deficiency state, and the striking improvement and relief of symptoms following the second and third courses of radiation therapy. The intercurrent *Necator americanus* infestation may be responsible for the eosinophilia demonstrated.

Case II. F. L. This white male, aged three, of Mexican extraction, was admitted to John Sealy Hospital June 20, 1942, complaining of a tumor on the head which had appeared after a fall out of bed three months previously. Questioning of the parents revealed definite polyuria and polydipsia, which they had not noticed, present for the same length of time. The parents stated that this was their eighth child, and that he had developed normally, except for an attack of whooping cough complicated by pneumonia. Both parents had received irregular and inadequate antiluetic therapy.

Significant physical findings were only: (1) two soft, semifluctuant, non-tender masses, each about 4 cm. in diameter, in the right frontoparietal regions; (2) palpable defects in the calvarium, showing under-cut edges, beneath the soft tumor masses.

Significant laboratory findings were: (1) urine, specific gravity of 1.001 to 1.010; (2) four eosinophils per high power field; (3) anemia of 4,000,000 with 80 per cent hemoglobin; (4) 4 plus Kahn and Wassermann reactions.

During the hospital stay, intake and output averaged about 2,000 cc.

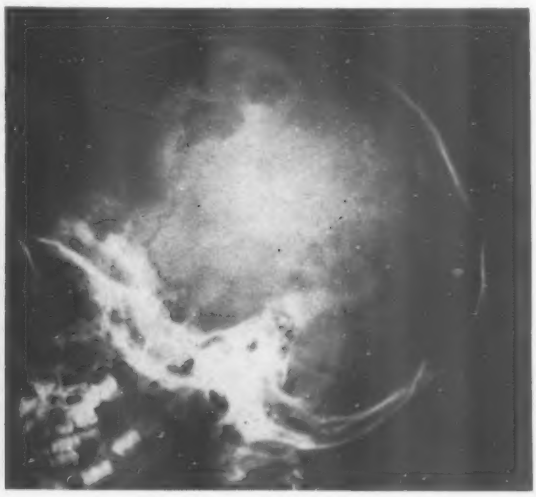

Fig. 4. Case II. July 3, 1942.

Fig. 5. Case II. December 10, 1944.

On December 18, 1942, biopsy of the cranial mass was reported by Dr. Paul Brindley, as follows: "There is a loose tissue of mononuclear cells intermixed with multinucleated giant cells. Practically all the cells show a foamy cytoplasm and in some the cytoplasm contains granules of light yellow pigment. The mononuclear cells are round, ovoid, and fusiform, with variable sized nuclei. Little or no mitosis is seen. Many cells are enormously distended with lipoid material, with crowding of their nuclei to the periphery. . . . This appears to be lipoid dystrophy, corresponding to Schüller-Christian disease."

Admission roentgenograms of the skull showed the two punched out defects in the right frontal and parietal bones as palpated (Fig. 4). The other bones showed no comparable change. Roentgen diagnosis of Schüller-Christian disease was made on the basis of the skull films, and the diabetes insipidus.

Between July 3 and July 10, 1942, a total of 900 r, measured in air, was given to each of two frontoparietal fields, 7 by 14 cm., at 152 kv. (peak), constant potential, 20 ma., 20 cm. skin target distance, using filtration of 1 mm. copper and 0.5 mm. aluminum, with a half-value layer of about 1 mm. copper.

During the hospital stay, intake and output remained at about 2,000 cc. after this course of therapy.

The patient remained in the hospital for some time, and between September 2 and September 16, 1942, he received another 1,200 r to each of the cranial fields, using the same technique. Roentgenograms taken at discharge, on October 3, showed slight new bone formation in the cranial defects. Additional roentgenograms taken on February 10, 1944 (Fig. 5), courtesy of Dr. B. M. Works, of Brownsville, Texas, showed practically complete filling in of the areas in the right frontoparietal region with new bone. Communication from the parents stated that the diabetes insipidus was very much improved, and there were no other new complaints.

Comment. This was the only patient in this series showing the defects confined to the cranial bones. The history of trauma in relation to the bony defects can be disregarded for obvious reasons. There were fewer signs and symptoms in this case than any of the others, yet the pathologist was unhesitant in confirming the roentgen diagnosis. The presence of congenital lues did not seem to interfere in any way with healing of the cranial lesions after radiation therapy.

Case III. D. A. This white female, aged four, was admitted to John Sealy Hospital September 12, 1945, with a chief complaint of "boils on the neck and head." The parents stated that these swellings had first appeared behind the ears three years previously. For one year, the swellings slowly increased in size, gradually becoming very painful to touch. They interfered with movements of the patient's head. There was no drainage.

The local physician gave an unknown amount of roentgen therapy to the mastoid areas, and the child returned to apparently normal health for a period of six months. At that time, the patient began to lose weight and become apathetic and showed no interest in play. She began to have difficulty rising from a sitting position or getting out of bed. Shortly thereafter, the generalized symptoms were spontaneously relieved, and peculiar tumor masses appeared over the left eye and in the left parietal region. The masses were soft, almost fluctuant, and non-tender. An unspecified amount of roentgen therapy was given with little or no improvement. Penicillin injection also produced no change.

One month before admission, worm infestation was found and the patient was given two courses of vermifuge.

Fig. 6. Case III.

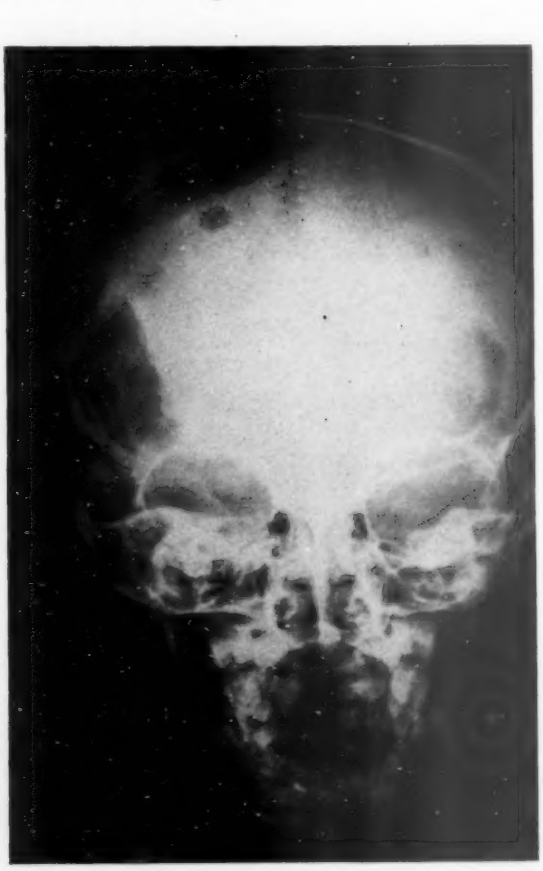

Fig. 7. Case III.

Significant physical findings were: (1) cachexia and emaciation; (2) head louse infestation; (3) drainage sinus in left parieto-occipital region; (4) generalized firm, non-tender lymphadenopathy; (5) 2 by 5 cm. soft lesion at base of skull; (6) depressed, round 3 cm. lesion left frontal bone; (7) draining sinus at vertex; (8) loose carious teeth; (9) systolic apical murmur.

Significant laboratory findings were: (1) anemia of 3,900,000 with 76 per cent hemoglobin; (2) leukocytosis of 9,700 with 2 per cent eosinophils; (3) bone marrow was somewhat hypercellular with relative increase in eosinophils and lymphocytes. No foam cells were seen.

Positive roentgen findings were: (1) Skull showed punched out typical lesions in both parietal bones, frontal bones, and occiput (Fig. 6 and 7). (2) Pelvis showed large punched out areas in the wing of each ilium. (3) The lumbar spine showed partial collapse of the first lumbar body, with marked condensation of bone (Fig. 8).

Biopsy diagnosis, on one of the parietal lesions, was "Eosinophilic granuloma, characterized by proliferation of large numbers of small round cells and a few multinucleated cells and large amounts of reticulin fibers."

Between October 4 and October 16, 1945, a total of 800 r, measured in air, was given to each of two parietotemporal fields, 10 by 10 cm., and a total of 1,000 r to each of two anterior pelvic fields, 10 by 15 cm. The factors were 217 kv. (peak), constant potential, 18 ma., 50 cm. skin target distance, using filtration of 0.75 mm. copper and 0.5 mm. aluminum, with a half-value layer of 1.2 mm. copper.

The patient remained in the hospital, showing only slight improvement.

Between November 26, and December 1, 1945, 600 r was given to each of two lateral lumbar fields, 10 by 10 cm., using the factors described above. Between January 7 and January 12, 1946, this last course was repeated, using the same factors.

Patient was discharged from the hospital January 12, 1946, and has not been seen since that time. Roentgenograms at the time of discharge showed no significant evidence of healing of any of the bone lesions.

Comment. This was one of the two patients who did not show diabetes insipidus as a presenting feature, but she had very widespread bone lesions, and was the only case showing involvement of a vertebral body. The course of the disease was undoubtedly altered to some extent by the unknown quantity of roentgen therapy given prior to our observation. Insufficient time has elapsed to evaluate the effect of adequate therapy. The biopsy diagnosis of eosinophilic granuloma is very interesting.

Fig. 8. Case III. Collapsed first lumbar body.

Case IV. F.N.H. This white male, aged four, was admitted to John Sealy Hospital July 27, 1939, with a chief complaint of fracture of the right humerus, present for three days. The parents stated that polydipsia and polyuria had been present for three years, and that for the same length of time they had noticed knots and depressed areas on his head. The right hip had been painful for about one year.

Two years previously, the patient had been under the care of Dr. H. H. Ogilvie, of San

Fig. 9. Case iv. Involvement of right humerus with pathological fracture.

Antonio, Texas, who had done a biopsy of a scalp lesion with the report "multiple myeloma of the bone." Aside from the scalp lesions and a destructive process in the left ninth rib and mandible, all found by roentgen examination, Dr. Ogilvie's findings were normal, except for an anemia of 3,330,000 with 41 per cent hemoglobin, and a leukocytosis of 15,850.

Scattered roentgen treatments were given between March 25 and June 11, 1937, using an unspecified amount of radiation. On July 26, 1937, the patient developed a staggering gait and a medial rotation of the left eye, and it was suggested that there was involvement of the vestibular apparatus. Blood count at this time showed slight improvement in the anemia, with 51 per cent hemoglobin, and showed 2 per cent eosinophils for the first time.

The patient was taken to the Mayo Clinic, where he was under the care of Dr. R. L. J. Kennedy and, during several admissions, received a large amount of roentgen therapy, with the last treatment in October, 1938. At the Mayo Clinic, the case was diagnosed as xanthomatosis.

There was nothing of significance in the other past or family history.

Three days before admission to John Sealy Hospital, the boy fell while playing and fractured his right humerus.

On admission, significant physical findings were: (1) fractured neck of right humerus; (2) marked malnutrition; (2) three depressed areas in calvarium; (4) marked exophthalmos; (5) small tumor outer canthus of right eye; (6) many teeth out, remaining ones loose.

Significant laboratory findings were: (1) urine, specific gravity of 1.005; (2) intake and output of around 2,300 cc. per day; (3) anemia of 3,600,000; (4) blood cholesterol of 222.

Roentgenograms of the entire skeleton showed cystic areas of destruction in the lower end of the left femur, both ends of the right tibia, in the skull, in the upper end of the right humerus, with a pathological fracture. Lesions were also seen in the upper end of the right femur, and in both innominate bones. The roentgen diagnosis was Schüller-Christian disease.

The patient was discharged from the hospital August 24, 1939, to receive radiation therapy as an out-patient.

Unfortunately our original roentgenograms of the skull were sent away for comparison and have been lost. Figure 9 was made during this admission.

Between September 20 and October 24, 1939,

Fig. 10. Case iv. September 9, 1940.

the patient was given 1,000 r to the right orbit, 750 r to the upper end of the right tibia, 750 r to the lower end of the left femur, 600 r to the right humerus, and 500 r to each of two parieto-occipital fields. The field sizes varied, all dosage was measured in air, and the radiation was given at 200 kv. (peak) (mechanically rectified), 5 ma., and 50 cm. skin target distance, using filtration of 0.5 mm. copper and 1 mm. aluminum. Between November 22, 1939, and January 29, 1940, the patient was given 1,000 r to the right orbit, 750 r to the right upper femur, and 1,150 r to the left upper femur, using the same factors. May 7 to 14, 1940, an additional 750 r was given to the left parietal bone, and

Fig. 11. Case iv. Roentgenogram of pelvis September 9, 1940, showing multiple punched-out lesions.

August 5 to August 15, 1940, an additional 800 r was given to a single occipital field.

Patient was readmitted to John Sealy Hospital on September 9, 1940, complaining of pain in the back and epigastrium, radiating to the left shoulder, present for three days, with epigastric swelling and frequent epistaxis. Figure 10 shows the skull at this time, Figure 11 the pelvis, and Figure 12 the right tibia.

Physical examination showed significant findings of: (1) malnutrition; (2) moderate icterus; (3) depressed areas in the skull; (4) loss of many teeth and the right lower alveolar ridge; (5) generalized lymphadenopathy; (6) a reddish-yellow scaly plaque, 2 by 3 cm., on the posterior aspect of the right shoulder.

The pain slowly subsided and patient was discharged October 14, 1940, without additional radiation therapy.

The patient was seen periodically as an out-

Fig. 12. Case iv. Roentgenogram of right tibia, September 9, 1940, showing solitary lesion.

patient, and for four months all of the symptoms were markedly alleviated.

The patient returned on February 7, 1941, with a marked increase in intake-output figures, to around 4,000 cc. He was treated as an outpatient, and between February 7 and April 9, 1941, received a total of 350 r to the right parietal bone, 600 r to the left parietal bone, 600 r to the right frontal bone, 400 r to each side of the occiput, 400 r to the right upper humerus, and 400 r to the region of the left hip. The same factors stated above were used.

The patient was readmitted to the hospital on November 3, 1941, with complaints of con-

Fig. 13. Case iv. November 3, 1941. Cranial lesions unchanged or larger.

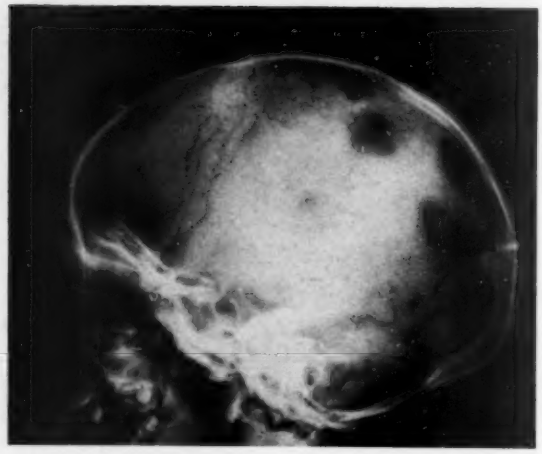

Fig. 14. Case iv. January 25, 1946.

tinued epistaxis, slightly increased diabetes insipidus, and persistence of the plaque on the right shoulder. The exophthalmos and the areas in the skull were considered to be unchanged (Fig. 13).

Significant laboratory findings were: (1) 5,000,000 red blood cells; (2) 3 per cent eosinophils in the white blood cell count (differential); (3) occult blood in the stools; (4) blood cholesterol of 267.

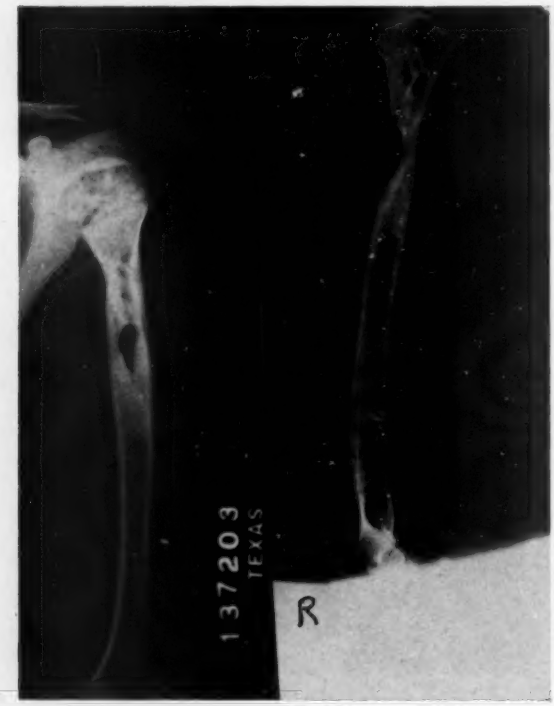

Fig. 15. Case iv. Right humerus, January 25, 1946.

The patient was discharged without treatment.

The next return to the clinic was on July 22, 1943, at which time there was a traumatic fracture of the right clavicle, and no other new complaints. The plaque on the right shoulder had disappeared, and the fractured right humerus was well healed.

Between July 26 and August 6, 1943, he was given a total of 450 r to each of the temporoparietal regions, using 217 kv. (peak), 18 ma., 50 cm. skin target distance, and filtration of 1 mm. copper and 0.5 mm. aluminum.

The next hospital admission was between

Fig. 16. Case iv. Pelvis, January 25, 1946.

November 28 and December 8, 1945, with complaint of pain in the lumbosacral region and left hip, increased by motion. Roentgenograms showed no new pathological changes in the region. There was a slight anemia of 4,600,000 and 2 per cent eosinophils were found in the differential count. There was a faint icteric tint to the skin, with a slightly positive direct Van den Berg reaction, and icterus index of 7.6. It was decided to run a vitamin A absorption test, and this was done by the courtesy of the Strong Memorial Hospital, Rochester, N. Y. The patient was given 7,000 International Units of vitamin A per kilo for a total of 157,000 units, and the vitamin A content of the blood serum was measured at four and seven hours, as well as the content before administration of the vitamin. The fasting sample showed a value of

10 (normal about 25)—at four hours, the value was 532 (normal about 250)—and at seven hours the value was 61 (normal about 140). The test seemed to indicate a vitamin A deficiency, with the vitamin being removed from the circulating blood more rapidly than normal.

The most recent hospital admission was between January 17 and January 25, 1946. There were no new complaints or findings, the differential count showed 5 per cent eosinophils, and there was a slight anemia of 4,550,000. On January 17 and 19, the patient was given 500,000 units of vitamin A by mouth. Roentgen examination of all the bones showed little or no change from the previous study (Fig. 14, 15 and 16).

Comment. This most spectacular patient has, over ten years of observation, exhibited every finding that has been described for Hand-Schüller-Christian disease. He illustrates very clearly the occurrence of spontaneous remissions, with no new bone lesions in the past five years. His growth has been markedly retarded and his present appearance is that of a withered and shrunken old man.

The massive amount of radiation has been given quite irregularly because of difficulty in following the patient, as the family is migrant. Perhaps the irregularity of the irradiation is the reason for only slight healing of the skull lesions over a period of years.

Mentally this patient is far above average. His intake and output seem fairly stationary at about 4,000 cc. It is to be hoped that significant improvement will follow the massive dosage of vitamin A. He will be followed as long as possible.

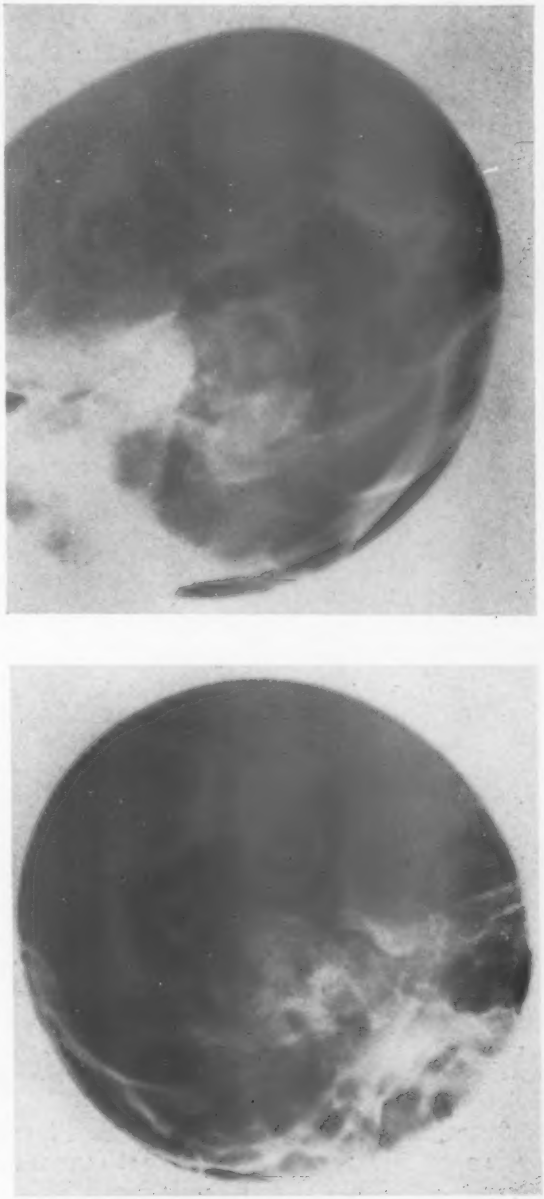

Fig. 17 and 18. Case v. March 23, 1942.

Case v. E.L., female, white, aged nineteen, unmarried, was admitted to John Sealy Hospital on March 21, 1942, with the chief complaints of deafness and amenorrhea, stating that she had never menstruated.

She had been under the care of her local doctor for three years, for excessive urination. A diagnosis of diabetes insipidus had been made, and the condition was well controlled with intranasal application of Armour's pituitary powder.

In August, 1941, she had an acute attack of bilateral mastoiditis, and a bilateral simple mastoidectomy was performed. No typical destructive process with suppuration was found, but there was extensive destruction of the mastoid processes by a peculiar grayish-red tissue.

Significant physical findings at admission were: (1) bilateral deafness; (2) infantile breast development; (3) absence of axillary and pubic hair; (4) bilateral mastoid scars; (5) infantile development of cervix and uterus; (6) dizziness and positive Romberg sign.

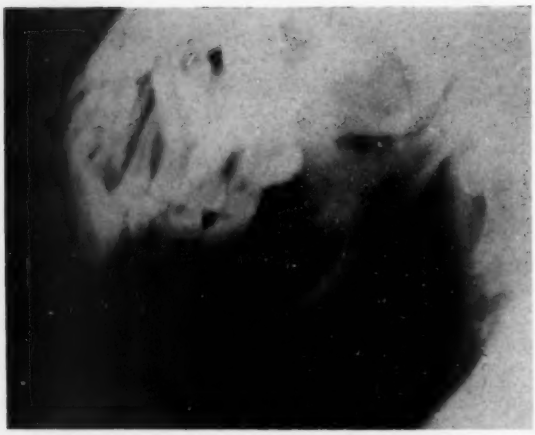

Fig. 19 and 20. Case v. June 30, 1942. Note extensive mandibular destruction.

The only significant laboratory finding at admission was a basal metabolic rate 20 per cent below normal.

Roentgenograms of the skull, taken March 23, 1942 (Fig. 17 and 18), showed extensive destruction in both mastoid processes, with extension to the temporal and parietal bones above and behind. There was seen to be extensive bone infection beyond the limits of destruction. Extensive bone erosion was also seen at the angles of both mandibles.

Roentgenograms of the long bones showed slight retardation of epiphyseal development, but no evidence of localized destructive lesions. Roentgenograms of the teeth showed a few areas of deep pyorrheal erosion. Spinal encephalogram on May 1, 1942, showed good filling of the ventricular system, and no significant deviation from a normal appearance.

Secondary mastoidectomy was performed on May 17, 1942, with an uneventful convalescence. The same peculiar appearing tissue was found, the pathological report being as follows: "Section of bone, the marrow of which is infiltrated by a number of fibroblasts and large cells with voluminous vesicular cytoplasm. There is little myeloid tissue, and a few lymphocytes, neutrophils, and plasma cells are present."

Patient went home for two weeks, and returned to the hospital June 30, 1942, remaining until July 19, 1942.

During this stay, a revision of the mastoidectomy and a sequestrectomy of necrotic bone from the mastoid areas were done; many teeth became loose, and several were lost. Roentgenograms of the mandible (Fig. 19 and 20) showed extensive destruction of the alveolar ridge of the mandible, bilaterally, with only a thin strip of the lower border of the mandible remaining.

On the next return, patient was in the hospital from July 26 to August 15, 1942, and the roentgenologist finally arrived at a diagnosis of a Schüller-Christian-like syndrome, on the basis of the bone destruction in mandible and skull, loosened teeth, diabetes insipidus, and the pituitary lesion evidenced by the presence of Froehlich's syndrome.

Between August 4 and August 15, 1942, she was given a total of 1,500 r, measured in air, to each of two temporoparietal fields, including the mastoid regions and mandible, each 10 by 10 cm., at 217 kv. (peak), constant potential, 18 ma., and 50 cm. skin target distance, using filtration of 1 mm. copper and 0.5 mm. aluminum. After discharge, several letters stated that she was doing better than she had done for over a year.

She was readmitted on September 30, 1944, with the chief complaint that, for the past two weeks, her skin had been turning yellow, the stools had been clay-colored, the urine was a dark smoky color, and there had been intense widespread pruritus.

Significant physical findings were: (1) Intense jaundice of skin and conjunctivae; (2) all the previous findings.

Significant laboratory data were: (1) eosinophilia of 11 per cent in the differential count; (2) bilirubinuria; (3) acholic stolls; (4) icterus index of 140; (5) direct Van den Berg with quantitative of 48 mg. per cent; (6) blood cholesterol of 278.

The previous course of irradiation to the

skull and mandible was repeated between October 8 and October 17, 1942. Supportive therapy with parenteral fluids and vitamin K showed no improvement in the deepening jaundice.

On November 2, 1942, the icterus index was 145 and the blood cholesterol was 345. On November 5, 1942, the icterus index was 178 and the blood cholesterol value was 480.

On November 27, 1942, exploratory laparotomy by Dr. E. J. Poth showed a large tumor of the head of the pancreas, completely obstructing the flow of bile. Biopsy of the pancreas was reported as showing no evidence of malignancy, only chronic pancreatitis and chronic hyperplastic lymphadenitis. Before closure, a cholecystojejunostomy and jejunojejunostomy were done in an effort to sidetrack the bile flow.

Postoperative course was slowly retrogressive, and, in spite of chemotherapy and supporting treatment, death occurred on December 14, 1942.

No autopsy was obtained.

Comment. This patient showed findings very similar to those of Schüller's first case, except for exophthalmos. Her disease had apparently originated about the time of puberty, giving rise to Froehlich's syndrome, and had progressed only very slowly till the terminal period of nine months, during which it became subacute. The obstructive jaundice, due to benign proliferation in the head of the pancreas, puzzled all observers both before and during the laparotomy. This was the only patient in this series to show a significantly elevated blood cholesterol value, and then only after biliary obstruction was practically complete. The author feels remiss that he did not make a correct diagnosis on the first roentgen examination of mastoids and mandible.

This patient showed less response to radiation therapy than any member of the group. It would have been desirable to irradiate the region of the head of the pancreas, after exploration, but opportunity for this was not available. It is regrettable that necropsy was omitted.

Case VI. This female, white, aged thirty-seven, married, was admitted to John Sealy Hospital August 11, 1942, partially comatose and disoriented. The family stated that she had first noticed left frontal headaches, which were steady and persistent, two years before, at which time she was pregnant. These persisted for about six months till the spontaneous deliver of her fourth child. At the time of this spontaneous delivery, patient had a convulsion, and the left eye suddenly became proptosed. Following this, there were recurrent convulsions at frequent intervals, and the patient became progressively disoriented, somnolent, and finally almost comatose. For three months before admission, there was complete incontinence of urine and feces. The family doctor told the family that she was crazy and advised them to bring her for hospitalization.

On admission, significant physical findings were: (1) coma; (2) incontinence of urine and feces; (3) left exophthalmos; (4) flexion contractions of the extremities.

Significant laboratory findings were: (1) anemia of 4,000,000; (2) 4 per cent eosinophils in the differential count; (3) blood cholesterol of 182.

Roentgenograms of the skull showed characteristic punched-out defects on both sides of the frontal bone, in the midline, with erosion of the roof of the left orbit. There were also large areas of erosion about the roots of the molar and premolar teeth (Fig. 21 and 22). A diagnosis of xanthoma of the frontal bones and mandible, of the Schüller-Christian type, was made.

Between August 17 and August 28, 1942, a total of 1,500 r, measured in air, was given to each of two 10 by 10 cm. frontal fields, at 217 kv. (peak), constant potential, 18 ma. and 50 cm. skin target distance, using filtration of 1 mm. copper and 0.5 mm. aluminum. During the course of treatment, the patient showed rapidly advancing clinical improvement, with orientation, return of sphincter control, and assumption of an interest in things about her.

It was decided, after the improvement had become stationary to do an exploratory operation and establish a definite diagnosis. On September 24, 1942, Dr. S. R. Snodgrass did a left frontal craniotomy, exposing a film of pinkish-brown tumor in the subdural space, extending to the midline, fusing with the falx, invading a tributary vein of the sagittal sinus, extending down along the anterior and inferior surfaces of the left frontal lobe, with a large dense mass on the roof of the left orbit. Biopsy specimen was taken, and closure effected.

Biopsy report, by Dr. Paul Brindley, was:

Fig. 21 and 22. Case VI. August 11, 1942. Extensive defects in the frontal bone and mandibular absorption.

"Very cellular tissue with reticular ground substance. Some mitoses. Cells are very pleomorphic. Small cells with round nuclei predominate. Some fresh blood and scanty reticulum (solid tumor mass). Other sections show a membrane with papilliform folds lined with cells. Within the membrane there are fibroblastic cells as well as many of the small cells with round nuclei. Reticulin stain shows abundant fibrils. Vessels are fairly frequent. Conclusion: Diffuse cellular tumor, growing fairly rapidly. No definite classification of the histogenesis of this growth."

The postoperative course was uneventful, with continuing general improvement.

Between October 5 and 17, 1942, an additional 1,500 r, measured in air, was given to each of the two frontal fields, using the factors described above.

The patient remained at home till June 8, 1943, living a relatively normal life, able to do her own housework, but during the interval she noticed occasional right frontal headaches and slowly increasing polydipsia and polyuria.

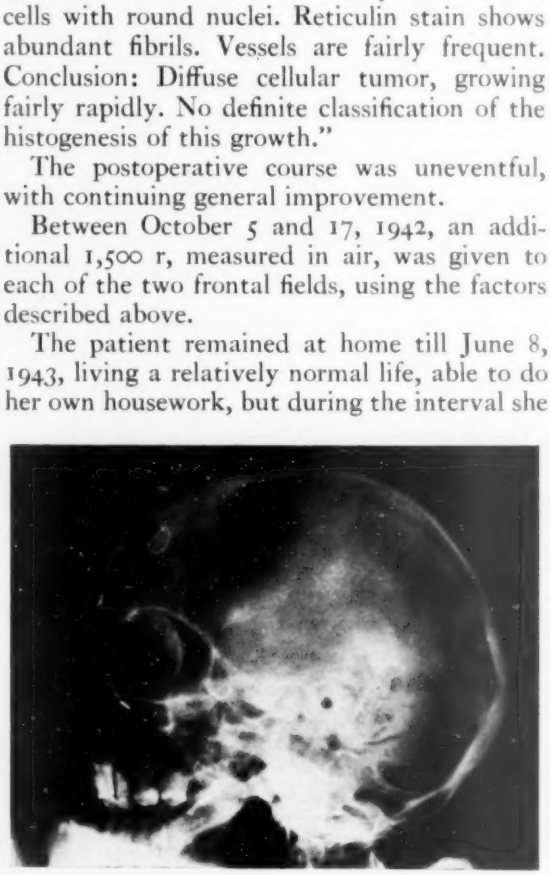

Fig. 23. Case VI. June 8, 1943. Note extension and large frontal flap.

On return to the hospital, June 8, 1943, roentgenograms of the skull showed extension of the cranial lesions (Fig. 23), and another course of roentgen therapy was given, with 1,900 r, measured in air, to 6 by 8 cm. fields, right, left, and anterior, over the involved regions of the skull, using the factors described above. Patient went home June 25, 1943, and returned August 11, 1943.

One week before return, she became nauseated and bled very extensively from her gums.

On readmission, significant physical findings were: (1) intense jaundice; (2) diffuse petechial hemorrhages and ecchymoses; (3) roentgen-ray discoloration over frontal regions; (4) left exophthalmos; (5) bleeding spongy gums; (6) blood pressure of 96/58.

Significant laboratory findings were: (1) anemia of 2,200,000; (2) white blood cell count of 2,200; (3) slight albuminuria; (4) urine sediment showed 8 red cells per high power field; (5) platelet count 26,160; (6) greatly increased bleeding, clotting, and prothrombin time.

Little except supportive therapy was given and the patient rapidly became worse and died on August 15, 1943.

Comment. The most striking diagnostic features of this patient were the convulsions and somnolence, due to the very extensive leptomeningeal spread over the frontal lobes, first interpreted as a postpartum psychosis. After radiation therapy and decompression, this patient's cranial symptoms were relieved for a year to the extent that she was able to lead a fairly normal life, though there was no change in her unusually extreme exophthalmos. The biopsy description of reticulo-endothelial hyperplasia, without foam cell formation, confused everyone connected with the case, though all were most impressed with the striking symptomatic improvement after radiation therapy. The terminal development of an acute hemorrhagic diathesis closely resembles the course of the recorded cases of Letterer-Siwe disease. The terminal jaundice suggested involvement of the liver as well as the hemopoietic system. Unfortunately, necropsy permission was not obtained.

Case vii. E.S., female, Negro, aged fifty-three, married, was admitted to John Sealy Hospital, March 4, 1944, for a work-up as a patient with chronic cholecystitis. She gave a history of inflamed eyelids and sinus trouble for twenty years, and attacks resembling gallbladder colic for one year.

Significant physical findings were: (1) tenderness over gallbladder region; (2) chronic pansinusitis; (3) chronic conjunctivitis and blepharitis; (4) obesity; (5) blood pressure of 198/80; (6) limitation of motion of cervical and dorsal spine; (7) multiple papulosquamous nevi over the back, slightly darker than the skin, diagnosed by Dr. C. N. Frazier as xanthomata; (8) mild exophthalmos, bilateral.

Roentgen findings were: (1) normally functioning gallbladder; (2) advanced arteriosclerotic disease with expansile pulsation of the aortic root; (3) marked osteoarthritis of the cervicodorsal spine; (4) a punched out lesion, 4 by 5 cm., in the left temporoparietal region (Fig. 24); (5) marked erosion about the roots of all the teeth.

Significant laboratory findings were: (1) electrocardiogram showed advanced myocardial damage; (2) anemia of 4,000,000; (3) differential count showed 4 per cent eosinophils; (4) blood cholesterol of 210.

In view of the multiplicity of complaints and the shortage of hospital beds, patient was discharged without treatment, and no follow-up has been obtained.

Fig. 24. Case vii. March 4, 1944. Large left temporoparietal defect.

Comment. This patient, though inadequately studied, showed, in addition to cranial and mandibular defects, the only instance I have been able to find of cutaneous involvement of reticulo-endotheliosis in the Negro race. The sinusitis and conjunctivitis were her most outstanding complaints, once a normal gallbladder had been demonstrated.

Discussion

The most important single feature in diagnosis of reticulo-endotheliosis is the appearance of the cranial bones on roentgen examination. The characteristic lesions show only bone destruction without evidence of any new bone formation, have sharp edges which may be irregular, and produce a larger defect in the inner than

the outer table. These findings are due to slowly increasing size of individual lesions, with destruction of adjacent bone by pressure. The lesions, when palpable, are soft, which readily distinguishes them from malignant metastases, but most commonly the scalp shows areas of depression at the site of the lesions. The lesions in other bones closely simulate the benign bone cyst.

Diagnosis is based on the presence, together with characteristic defects in the cranial bones, of malnutrition, chronic low grade anemia, disturbances in growth and development, diabetes insipidus, involvement of other bones, especially the mandible, and less frequently exophthalmos, jaundice, skin lesions, generalized lymphadenopathy, aural discharge, hemorrhagic diathesis and occasionally pituitary-gonad disturbances.

The histopathological picture is so variable that pathological confirmation of the diagnosis is the exception rather than the rule—a single specimen examined by a number of pathologists will frequently be given as many different diagnoses.

The important feature of management of reticulo-endotheliosis appears to be early recognition of the disease, through astute correlation of the bizarre findings with the characteristic appearance of the cranial roentgenograms. As soon as the diagnosis is made, radiation therapy to every organ or region which the signs indicate is involved is highly desirable. One of the author's patients (Case IV) has been carried for ten years in this manner.

The disease, except in the acute Letterer-Siwe type, shows a strong tendency toward spontaneous remission. Irradiation is given in the hope of supporting the patient and causing retrogression of individual lesions and improving the patient symptomatically till the appearance of a remission. Nutritional therapy, of a number of kinds, has not been shown to be of value, though we are awaiting the result of massive doses of vitamin A in our Case IV.

Radiation therapy may be given in divided doses, to any involved area, using filtration sufficient to protect the superficial structures and insure adequate depth dosage. Deep roentgen therapy, in the 200 kv. range is entirely adequate for this purpose. Dosage to an individual lesion or region is much smaller than the cancerocidal dose would be, and adequate treatment can usually be given without permanent change in the skin or its appendages.

All of the cases, except one, which I have treated, showed very marked improvement after irradiation of the involved areas—the exception showed some improvement but no therapy was directed to the pancreas, site of the tumor causing her terminal symtoms. In each of the 4 children, therapy was apparently of definite supportive value until appearance of a spontaneous remission.

In infants and very young children, the form of reticulo-endotheliosis known as Letterer-Siwe disease is acute and rapidly fatal. Also, in adults, reticulo-endotheliosis seems to run a chronic to subacute course, with massive terminal involvement of an essential organ or system. However, the great majority of cases apparently develop in children beyond infancy and before puberty. In these, local radiation therapy of involved areas has been shown to be of great value and to improve the prognosis, both immediate and final, very markedly.

CONCLUSIONS

1. The literature on reticulo-endotheliosis is reviewed at length.

2. Evidence is presented that reticulo-endotheliosis may assume an acute, fatal, non-lipoid form, in infants and young children, known as Letterer-Siwe disease.

3. In older children and adults, the less acute disease is characterized by lipoid deposit and corresponds in every respect with what has been described as Hand-Schüller-Christian disease.

4. At any stage of its course, Hand-Schüller-Christian disease may assume a subacute fatal character.

5. Evidence is presented that so-called

eosinophilic granuloma is a variant form of reticulo-endotheliosis, with a usual solitary bone lesion, eosinophilia in the areas of reticulo-endothelial proliferation, and probable widespread soft tissue involvement.

6. The small amount of reported experimental work in reticulo-endotheliosis is described.

7. The bizarre and widely varied presenting signs of reticulo-endotheliosis are shown to be due to largely accidental location of areas of reticulo-endothelial proliferation.

8. Seven cases of reticulo-endotheliosis are presented, with histories and illustrations, and are discussed at length.

9. The importance of roentgenography of the skull in diagnosis is emphasized.

10. The role of radiation therapy in palliation of the patient until the appearance of a characteristic spontaneous remission in the non-acute type is emphasized.

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NODULAR PULMONARY DENSITIES DUE TO SCARS OF MULTIPLE PULMONARY INFARCTS*

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THE roentgenographic appearance of pulmonary infarcts has recently been studied extensively by Hampton and Castleman.[1] Their studies, with pathologic anatomical correlation, show that pulmonary infarcts are rarely triangular in shape though their shadows may be roughly triangular. These authors state that the infarcts are always peripheral and sharp in outline.

One might suspect that a greater proportion of infarcts would present a triangular shadow, if the distal boundary thereof were limited to one plane of the visceral pleura. This would apply to smaller infarcts, which would have less chance of involving more than one surface, by geometric probability. It is also obvious that the direction of the central roentgen ray, in respect to the axis of symmetry, greatly influences the shadow outline.

Infracts are usually seen singly or in small numbers. When multiple, they are often large and associated with fatal outcome. However, they are occasionally seen as multiple small lesions, nodular or even miliary in size. The case reported herein is an instance of this type.

CASE REPORT

A forty year old Negro porter entered the San Francisco City and County Hospital on February 19, 1946, and died eight days later. He had "chills and fever" for two weeks and had felt ill with headache and a dull mid-abdominal pain. His urine had become foul smelling, he had nocturia, frequency and urgency. Exertional dyspnea had been present for six years, a slightly productive cough for two years and orthopnea for ten days before entry.

A previous hospital entry had been made in 1943 for tonsillitis. A roentgenogram of the chest on November 26, 1943 (Fig. 1) disclosed small nodular densities throughout both lung fields. The blood Wassermann reaction was positive and antiluetic therapy was given for one year. The blood pressure was 125/75. He had used "opium" intravenously for eighteen years, with chills frequently following injections.

At the last entry he appeared ill and dyspneic. He was shaking with chills. Temperature, 104.4° F.; pulse, 120 per minute; respiration, 38 per minute; blood pressure, 120/40.

The antecubital veins were scarred. Petechiae were noted in the right conjunctiva. The neck veins were distended and marked venous pulsations were visible with the patient in a sitting position. Moist rales were heard over the posterior position of the chest at the lung bases and wheezes throughout the chest. A violent cardiac apex impulse was felt in the sixth intercostal space 2 cm. lateral to the mid-clavicular line. There was an apical systolic thrill. The aortic second sound was absent. A loud to-and-fro murmur could be heard throughout the precordium. The spleen was palpable 3 cm. below the left costal margin. The liver was not palpable. Tenderness was present in the right costovertebral angle. The fingernails were slightly rounded and the feet puffy without pitting edema.

Blood: Hemoglobin 78 per cent (Sahli). Erythrocytes, 3,700,000; leukocytes, 11,800; neutrophil leukocytes, 82 per cent. Blood urea, 33 milligrams per cent. Serum proteins, 5.8 grams per cent (albumin 2.9 grams, globulin 2.9 grams). Blood Wassermann reaction, positive. The blood cultures showed gram-negative rods of the coli-aerogenes group. Urine: (twenty-four hour period) amount 900 cc.; specific gravity, 1.015; pH, 5.0; granular casts, 200,000; erythrocytes, 48,000,000; leukocytes, 72,000,000. Protein or sugar was not found.

Lumbar puncture gave no positive findings. Chest roentgenogram on February 19, 1946, showed "a small triangular density at the right costophrenic angle" plus the small nodular densities noted three years before (Fig. 2, 3).

He was given sulfadiazine for the bacteriemia and digitalis for cardiac decompensation. How-

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ever, dyspnea, leg edema and the number of petechiae increased. Irrationality and coma progressed and he died eight days after entering the hospital.

Postmortem examination revealed aortic valve vegetations with rupture of the posterior cusp. The liver and spleen were large. Numerous septic infarcts were present in the spleen and kidneys. These were of recent origin. In addition, there were quite numerous small (2 to 5 mm.) firm nodules throughout both lungs. Histopathologic studies, using elastic tissue stains and the usual hematoxylin-eosin stains, showed these nodules to have characteristics of old infarcts without evidence of inflammatory

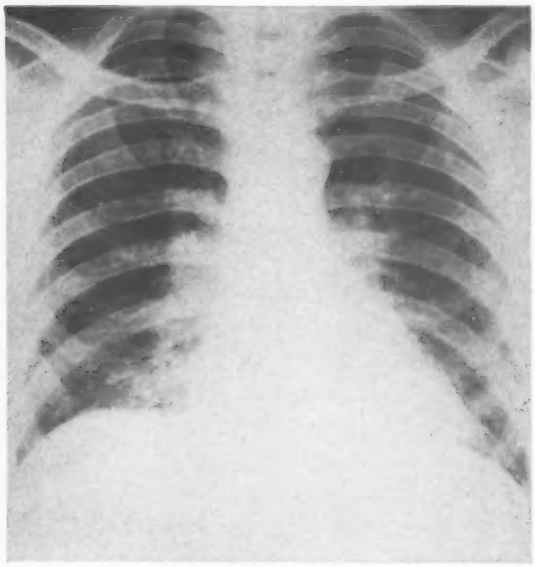

Fig. 1. Fine densities throughout both lung fields in 1943.

reaction. The following is a description of one of these by Dr. David A. Wood:

6D-52 F: "One section of lung shows a small oval non-encapsulated nodule which consists of collapsed pulmonary tissue in which all vestiges of alveolar walls are absent. Present in the condensed fibrous tissue are occasional small capillaries lined with swollen endothelium and containing blood. There is no inflammatory reaction in this nodule. Elastic tissue stains show the usual amount of elastic tissue fibers. Otherwise the pulmonary tissue shows moderate deposition of anthracotic pigment in the septa and some of the alveolar walls. Most of the alveoli in the adjacent parenchyma are air-containing. Some alveoli contain desquamated cells and

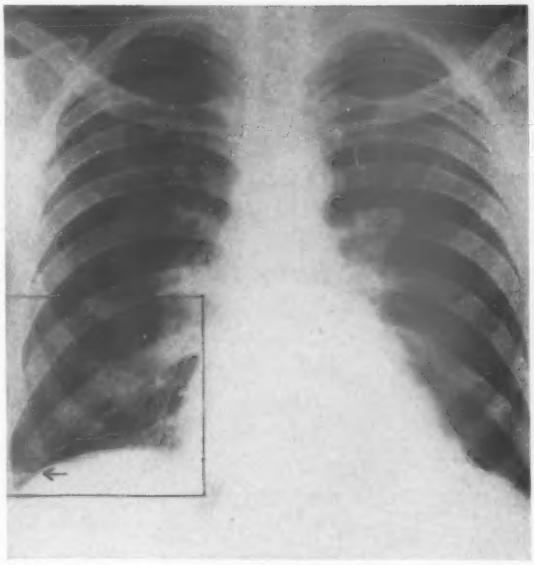

Fig. 2. Appearance of lungs in 1946 similar to 1943.

slightly thickened alveolar walls. A few of the latter are infiltrated with lymphocytes."

COMMENT

The three year persistence of the scattered pulmonary nodules and the eventual appearance of one triangular density in the final fulminating episode of bacteriemia

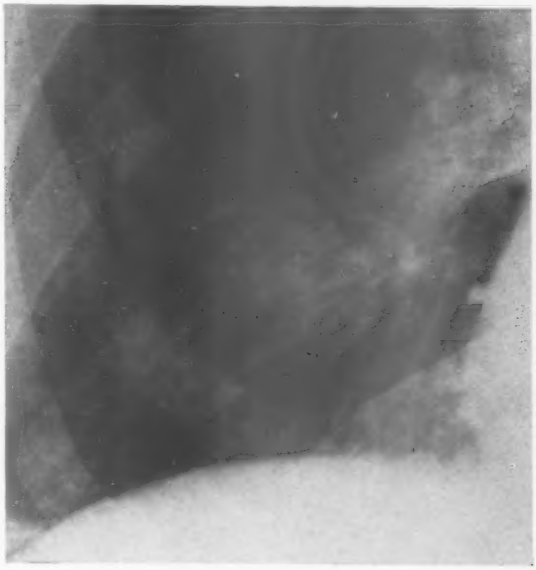

Fig. 3. Right lower lung field in 1946. Note the triangular clouding and profuse fine nodular densities.

might have assisted in the identification of the pulmonary process. The triangular shape of the largest pulmonary infarct corresponded to the usual didactic description. However, the true significance of the multiple nodular densities was not recognized.

Whether these old infarcts were secondary to bacterial emboli or injected particulate matter incidental to his addiction is still undetermined. The lack of evidence of inflammatory reaction or scarring is of interest to note. Obviously the recent infarcts were associated with the endocarditis and bacteriemia. That intravenous use of opiates by addicts is not infrequently followed by bacteriemia and multiple septic emboli has been reported.[2] In this instance, there may well have been some insoluble residuum of crude or poorly refined opium obtained in more recent years.

It is recognized that there is, unfortunately, no diagnostic feature which would permit recognition of this unusual condition in the roentgenogram. The term "infarctosis" was applied to this unusual roentgenographic appearance by L. H. Garland following his review of the case. This seems a most descriptive and apt designation.

SUMMARY

A case of multiple pulmonary infarcts, simulating disseminated or miliary pulmonary disease, is presented. This occurred in an addict using opiates administered intravenously for eighteen years.

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THE CLINICAL AND ROENTGEN MANIFESTATIONS OF PNEUMONIA DUE TO *BACILLUS MUCOSUS CAPSULATUS* (PRIMARY FRIEDLÄNDER PNEUMONIA)*

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INTRODUCTION

FRIEDLÄNDER pneumonia is a relatively infrequent yet very important type of pneumonia. It is a severe disease in which the mortality is markedly elevated,[3,32] approximately 70 to 80 per cent of cases terminating fatally. In the patients who do not succumb, there is a high incidence of complications, many developing chronic lung abscesses which run a very prolonged course. The use of sulfonamides[20,30] and penicillin[10,36] has not influenced the mortality in Friedländer pneumonia to an appreciable extent. However, the early administration of streptomycin[16,24,27] has resulted in recovery in some instances with a definite diminution in the death rate. As the ordinary types of pneumonia have been greatly reduced in frequency and mortality by modern methods of therapy, the more virulent and resistant Friedländer pneumonia has become of increasing importance. Early recognition of the disease and the immediate institution of therapy are of the utmost importance. The roentgenologist's ability to indicate the diagnosis or to confirm the suspicions of the clinician may prove of the greatest value. As with other rare and unusual conditions, the diagnosis will be made only after the demonstration of findings characteristic of the disease and if the possibility of the condition is borne in mind.

ETIOLOGY

Friedländer pneumonia is more common in males in the ratio of 5:1. The majority of cases occur between forty and sixty-five years of age, only a few instances having been recorded in children.[12,21,25] A history of alcoholism is often present and is probably a predisposing factor. Malnutrition and other debilitating influences are also significant. Cold and exposure are important, there being a definitely higher incidence in the winter months. There is frequently a history of long-standing, persistent cough or previous respiratory tract infection with bronchitis and bronchiectasis. It has been noted by many authors that a source of infection of the Friedländer bacillus is present in the accessory nasal sinuses, the throat, or upper air passages. In one of our patients, elimination of the organism from the sputum was not accomplished until the focus in the sinuses had been eliminated. In this type of case, there is not infrequently a history of previous attacks of Friedländer pneumonia.

BACTERIOLOGY AND EPIDEMIOLOGY

Friedländer's bacillus was first described in 1882 by Carl Friedländer[14] who considered it the causative organism in most cases of pneumonia. With the subsequent demonstration that the pneumococcus was the etiologic agent in lobar pneumonia,[34] the pendulum swung to the belief that Friedländer's bacillus was never the cause of pneumonia, being merely a secondary invader. It is now generally accepted that a small but definite percentage, variously estimated at from 0.5 to 5 per cent,[32] of all pneumonias are of the primary Friedländer type. Friedländer's bacillus, commonly referred to as *Bacillus mucosus capsulatus*, is a short, gram-negative, nonmotile, nonspore-forming rod with a thick capsule.[39] Julianelle,[22] in 1926 and 1930, identified two main types: A and B. Types

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C, D, E and a mixed group X are also described. The soluble specific substance of the capsule of Friedländer's bacillus Type B and of pneumococcus Type II are closely related and the sera of one will protect mice against infection with the other. Type specific antisera have been found useful in therapy. Friedländer's bacillus was demonstrated in the upper respiratory tract of 2.2 per cent of a large group of healthy individuals tested by Bullowa, Chess and Friedman.[6] The organism may be present in the blood stream and distant metastatic foci occur in the meninges, vertebrae, and so forth. In apparently healthy individuals Friedländer's bacillus has been isolated from the stools, urine, bile, vagina, and uterus, In addition it has been found in soil, air, dust, mud, and water.[2] The incubation period is not definitely known but appears short, being estimated at about forty-eight hours.[12,32] Epidemics are rare although they have been reported.[38] The role carriers play in this disease has not been determined.[5] Baehr *et al.*[2] believe that primary pulmonary Friedländer infection is rare and that most cases begin as liver, biliary tract, and urinary infections, the lung being a secondary seat of invasion, oftentimes as a terminal event. Study of the literature, however, indicates that definite primary infection does occur. It will be remembered that the pneumococcus, streptococcus and other pathogens similarly may appear as saprophytes, secondary invaders, or in association with extrapulmonary invaders, and Friedländer's bacillus undoubtedly is similar to the pneumococcus in this respect also.

PATHOLOGY

Any lobe or combination of lobes may be involved. Usually more than one lobe is affected.[20,28] The consolidation is oftentimes described as being lobular or "confluent lobular,"[23] rather than lobar. Solomon[32] reports lobar type of involvement in 84 per cent and lobular in 16 per cent. He noted involvement of more than one lobe in 65 per cent of his cases. There is apparently no predilection for any particular lobe. The affected lobe or lobes are usually bright red to chocolate-brown in color and less freqently pink or grayish. The involved portion of the lung is voluminous. There is considerable inflammatory reaction in the pleura with a reddish, fibrinous pleural exudate. The cut surface of the lung is covered with viscid, abundant, mucinous exudate which characteristically is very tenacious. The alveolar exudate is usually brick-red, glairy, mucoid, or gelatinous. Friedländer's bacillus is well known as a destroyer of parenchymal lung tissue.[17,19,28] Abscesses filled with pus are of common occurrence.[33,35] Destruction of the alveolar walls is often found microscopically although it may not be demonstrable grossly. Pulmonary edema, pleural effusion, empyema, pericarditis, and meningitis are complications found at autopsy in a high percentage of cases. Portal cirrhosis and fatty liver are probably not infrequent.[20] Jaundice is present in over 10 per cent of acute cases.[32]

CLINICAL COURSE

The onset is sudden, usually starting with cough, sputum, hemoptysis, pleuritis, pain, and a chill.[20] Cyanosis and dyspnea may be present. In many instances, the patient appears desperately ill from the onset of the disease. The temperature varies from 99° to 106° F., although most observers note that temperatures below 102° F. are common.[32] The pulse and respiratory rate correspond to the elevation of temperature. The leukocyte count may aid in suggesting the diagnosis as in many cases there is only a relatively slight leukocytosis.[28] Solomon[32] found no marked increase in the white count in 63 per cent of his cases, and in only 12 per cent did it exceed 20,000. Leukopenia and monocytosis may occur. The physical signs are not diagnostic. The sputum may be brick-red and homogeneous as though blood and mucus had been mixed together to form an emulsion. This appearance is found in only a small percentage of cases, the majority showing sputum similar to that found in

lobar pneumonia. Bacteremia is common but is usually not marked and there is no direct relationship between the mortality rate and the degree of bacteremia.[32] The usual course of acute Friedländer pneumonia is to early death, usually in two to six days, and this has been the end result in from 51 to 97 per cent[3,20,32] of previously reported cases. The average mortality rate is 70 to 80 per cent. Some patients recover rapidly and completely. The third possibility is the development of a chronic form of Friedländer pneumonia with necrosis and the formation of one or more thin-walled lung abscesses.[7,17,22,26] There is a definite tendency for tuberculosis to develop after Friedländer pneumonia.[20]

DIFFERENTIAL DIAGNOSIS

Acute Friedländer pneumonia must be differentiated from pneumonia caused by the pneumococcus, hemolytic streptococcus, staphylococcus, influenza bacillus and the viruses.[13] The individual with Friedländer pneumonia is usually over forty, males predominating greatly. Frequently there is a history of a chronic respiratory infection preceding the onset of the acute Friedländer pneumonia.[13,28] The patient while obviously gravely ill may have a temperature which does not rise above 102° F. Herpes, which is so commonly seen in lobar pneumonia, is not frequent. The sputum is usually thick, glairy, mucoid, and oftentimes brick-red. In many cases, the physical signs are not commensurate with the degree of lung involvement as found by roentgen examination or at postmortem. In pneumococcus pneumonia, the temperature is usually high and the physical signs of lobar consolidation are characteristic. The respirations are short and grunting and the sputum is rust colored. A well marked leukocytosis, predominantly polymorphonuclear cells, is present. Hemolytic streptococcus pneumonia often follows streptococcal infection elsewhere in the body. Pharyngitis, laryngitis, and tracheobronchitis commonly precede the pneumonia. The consolidation is of the diffuse, patchy type. The sputum is blood streaked or bloody. Staphylococcus pneumonias are usually seen in early childhood. In adults this type of pneumonia is metastatic from other foci or follows influenza. The temperature is high and the consolidation is patchy. The sputum is purulent and is usually mixed with varying amounts of blood. Influenzal pneumonia is most common in infancy and early childhood. In adults it occurs in pandemics as in 1918. It may develop in adults after long-standing chronic bronchopulmonary infections. The virus types of pneumonia are more common in young adults. The onset tends to be gradual with headache and a chilly sensation but rarely a true chill. Presternal soreness occurs frequently. True consolidation is uncommon. A peribronchial infiltration, usually spreading from the hila, is seen at roentgen examination. Weakness and persistent cough are frequent but severe prostration and death are rare. The chronic form of Friedländer pulmonary infection, usually with thin-walled cavities and abscesses, must be differentiated from the large group of diseases characterized by patchy or peribronchial infiltrations.[8,17,23] Pulmonary tuberculosis is usually the first consideration.[7,33,27] Some cases of Friedländer pneumonia, because of the roentgen demonstration of areas of marked, uniform density, may be diagnosed as neoplasm of the lung with fluid, atelectasis, or both. The history of acute onset, marked toxicity, leukopenia, and comparatively low temperature, coupled with the inability to obtain fluid on chest tap, are important factors in arriving at a correct diagnosis.

ROENTGEN FINDINGS

There is no single roentgen picture which occurs regularly in Friedländer pneumonia. Based on a review of the literature and the 14 cases which we have observed, the following roentgenologic classification of this disease is suggested:

Group 1. The Massive Lobar Consolidation Type. The affected portion of the lung is markedly and uniformly dense, apparently

due to very large quantities of exudate. At times, a single lobe is involved. In others, massive consolidation of the entire lung field occurs. These patients may be seen early in the course of the disease and at that time, the dense, lobar type of consolidation is fully established, apparently having been present almost from the onset. In some instances, the affected lobe(s) seem to bulge, fluid (the so-called "drowned lung") being suspected. The impression of fluid is further strengthened when the entire lung field is involved with displacement of the heart and trachea to the opposite side. The density obliterates the outlines of the ribs, the heart border and, when the base is involved, the shadow of the diaphragm and costophrenic angle. The roentgen changes are in practically every instance interpreted as being due to neoplasm and fluid and the chest is tapped without fluid being obtained. Massive atelectasis may be suspected, although the absence of ipsilateral displacement of the heart and mediastinal contents militates against this diagnosis. The cases which present the massive lobar consolidation are practically always rapidly fatal, death resulting from an overwhelming toxicity and in some instances occurring within twelve to twenty-four hours from the onset.

Group 2. The Lobular Consolidation Type. In the early stages, there is a patchy, irregular density which later develops into a confluent consolidation scattered through one or more lobes. This group is larger than the massive lobar variety and is usually indistinguishable roentgenographically from other lobular pneumonias. This type has a very high mortality rate also, although many recover with resolution or continue as the chronic form of Friedländer pneumonia to be described below.

Group 3. The Chronic Form Characterized by Lung Abscess Formation and Pulmonary Suppuration. As mentioned above, lung abscesses may form relatively early in the course of Friedländer pneumonia. They are apparently due to the thick, viscid, tenacious exudate which produces obstruction with subsequent atelectasis and ischemia. Delayed resolution is also a common occurrence with chronic suppuration and abscess formations of varying size which persist for very long periods. These changes closely resemble pulmonary tuberculosis or bronchiectasis both clinically and roentgenologically. They may undergo partial or complete clearing only to recur despite therapy. In some instances, the destruction of pulmonary tissue is so extensive that it is referred to as spontaneous lobectomy.

It is in the massive lobar consolidation (Group 1) that the roentgenologist may be of the greatest aid to the clinician. Massive consolidation involving one or more lobes should immediately suggest the possibility of Friedländer pneumonia. Since death may occur within a matter of hours or days, early roentgen diagnosis is of the utmost importance. Streptomycin appears effective in some instances, while penicillin and the sulfonamides are of doubtful value. Prompt institution of therapy may prove a life saving procedure. The diagnosis of chronic Friedländer pneumonia must be considered when the roentgenogram demonstrates diffuse, mottled, peribronchial infiltrations with cavitations, particularly in cases in which the tubercle bacillus cannot be isolated. Although Friedländer's bacillus may be only one of several organisms found the use of streptomycin, particularly by the aerosol method, may produce beneficial results.

REVIEW OF THE LITERATURE

Friedländer[14] first described the *Bacillus mucosus capsulatus* in 1882 and erroneously concluded that this organism was the etiologic agent in the majority of cases of lobar pneumonia. Etienne[11] in 1895 recorded early observations as to the prevalence of the Friedländer's bacillus in the respiratory tract of normal individuals and noted the patchy nature of the pulmonary lesion in type of pneumonia. Apelt[1] in 1908 reported a series of cases and stressed the high mortality as well as the tendency to the development of lung abscesses. Sisson and Thompson[31] listed 33 cases of primary Friedländer pneumonia in their

review of the literature prior to 1915 and added 4 additional cases. Zander[38] (1919) reported an epidemic of 411 cases in a labor camp in Germany. This is the largest number of cases recorded by any author and the only epidemic. However, a critical analysis by Solomon[32] casts doubt upon the authenticity of these cases as being definitely proved Friedländer pneumonia. Julianelle[22] in 1916 classified Friedländer's bacillus into three main types: A, B, and C, and a heterogenous group X by means of agglutination, absorption, and precipitin reactions and by passive protection tests. Belk[3] reported 18 cases, all fatal. Westmark[37] (1926) observed the tendency of Friedländer pulmonary infection to simulate tuberculosis. Kornblum[23] in 1928 and a year later with Collins[8] described the roentgen appearances of Friedländer pneumonia in both the acute and chronic forms. He pointed out the close similarity of the chronic forms to pulmonary tuberculosis and gave a roentgenologic classification of the stages of primary Friedländer pneumonia. Olcott[28] found that in 4 of 6 cases the consolidation at postmortem was lobar in character. He noted the occurrence of leukopenia and the high percentage of large mononuclear cells. Solomon[32] (1937 and 1940) reviewed both the primary and chronic forms of Friedländer pulmonary infection and included 32 cases of the primary type and 17 of the chronic variety. The acute cases were predominantly lobar (84 per cent), the remainder being bronchopneumonic in type. He indicated the frequency of multilobar involvement, the characteristic sputum, the leukopenia, and the high mortality rate. Solomon stated that the chronic form of Friedländer infection usually followed the acute disease and emphasized the frequency of upper lobe involvement with cavitation, findings which usually resulted in an incorrect diagnosis of pulmonary tuberculosis. Bullowa, Chess and Friedman[6] in 1937 recorded 41 cases of acute pneumonia due to the Friedländer bacillus. They quoted French authors who classified the various types as follows: (1) the hyperacute characterized by overwhelming toxicity, massive consolidation and practically always a fatal termination; (2) the acute type with hepatization, abscess formation, and occasional recovery; and (3) the subacute or suppurative pneumonic type in which the patient survives the first stage to experience an extended period of remission and exacerbation, ultimately developing clinical and roentgenographic evidence of cavitation. Bullowa observed that the consolidation was frequently lobar but noted the frequency of extension beyond the interlobar fissure with multilobar involvement. Perlman and Bullowa[30] in 1941 recorded 37 cases and stressed the inadequacy of therapy. Hyde and Hyde[20] in 1943 reported 51 cases. They found the incidence of Friedländer pneumonia to be 1.6 per cent of the pneumonias at Bellevue Hospital. All lobes showed approximately equal frequency of involvement. Positive blood cultures were present in 45 per cent of cases. The role of streptomycin and streptomycin with penicillin, which may be an effective form of therapy for Friedländer pneumonia, has been reviewed by Finland *et al.*[16,29], Bishop and Rasmussen,[4] Learner and Minnich[24], Welford,[36] Geier,[15] Nichols and Herrell[27], the Committee on Therapeutics,[9] Heilman,[18] and others.

CASE REPORTS

Case 1. The massive lobar consolidation type (Group 1). E. P., white male, aged sixty-three, was admitted October 17, 1946, with a history of pain in the left chest for the past week. The patient was acutely ill with labored breathing and moderate respiratory distress. The history could not be obtained from the patient and the family was vague in the description of the illness. There was a history of chronic cough and weight loss prior to the present illness. Temperature 100° F., pulse 118, respirations 35, blood pressure 140/70. On physical examination there was dullness over the left anterior chest and axilla with diminished breath sounds. Breath sounds were faintly audible at the left base posteriorly. The right lung was clear. Laboratory data: White blood cell count 16,400 (81 per cent polymorphonuclear cells); red blood cell count 3.5 million (60 per cent Hgb); nonprotein nitrogen 50 mg. The sputum was negative for

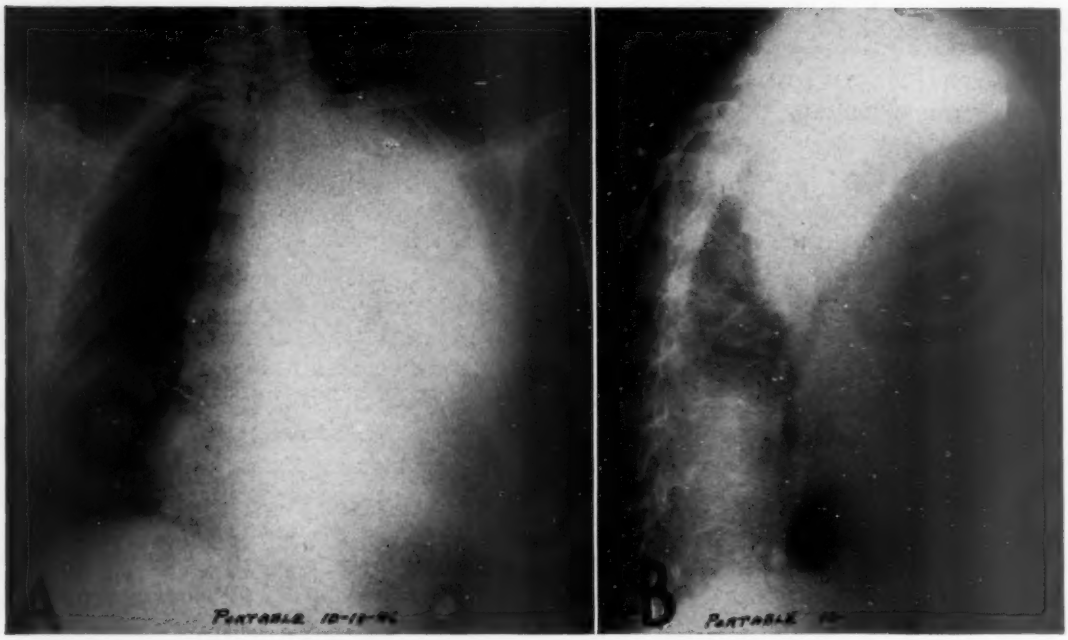

Fig. 1. Case 1. The massive lobar consolidation type of Friedländer pneumonia.

(*A*) Anteroposterior view one day after admission to the hospital. There is uniform density involving practically the entire left lung field except small areas at the apex and the axillary portion of the base. The density obliterates the outlines of the left ribs and cardiac border. The heart and trachea are displaced to the right. The left diaphragm is faintly visualized and is elevated.

(*B*) Lateral projection. The density involves the entire left upper lung field and the anterior portion of the middle and lower lung field, indicating involvement of the left upper lobe. The posterior portions of the left middle and lower lung fields show slight mottling, due to patchy consolidation of the left lower lobe. The left upper lobe is increased in size and bulges. The changes are those of massive consolidation of the left upper lobe, the uniform density being due to the great amounts of exudate. The affected lobe is large and bulges. The displacement of the heart and trachea to the opposite side produces a picture which closely simulates pleural effusion or tumor. At postmortem examination, the massive density of the left upper lobe was found to be caused by the thick, viscid exudate. (Courtesy of John F. Sheehan, M.D., St. Vincent's Hospital, Worcester, Mass.)

tubercle bacilli. The blood culture showed *Streptococcus viridans*.

Course. The patient's condition became steadily worse with increasing dyspnea. Treatment consisted of penicillin and oxygen. Repeated attempts were made to aspirate fluid from the left chest without success. The pulse remained approximately 100 and the respirations 30–40. His temperature never rose above 101° F. He died on October 23, six days following admission.

Postmortem. The left pleural cavity is obliterated by old and recent fibrinous adhesions. The heart and mediastinum are displaced slightly to the right. The left upper lobe is markedly increased in size, bulging in all directions. It is dark purple in color and completely solidified. On section it shows a reddish gray, finely granular appearing surface and there is a large quantity of thick pus exuding from it. The exudate was very viscid and gram stain showed large numbers of organisms morphologically typical of Friedländer's bacillus. The lower lobe also showed areas of consolidation, but this was much less marked than in the upper lobe. (This case is presented through the courtesy of Dr. John F. Sheehan, St. Vincent's Hospital, Worcester, Mass.)

Case II. The massive lobar consolidation type (Group 1). J. A., colored male, aged sixty-two, was admitted to the hospital March 14, 1946, in a very toxic condition. He stated he had been ill for four or five days with cough, left chest pain, severe chills, high fever, dyspnea, and profuse sweating. There was no history of

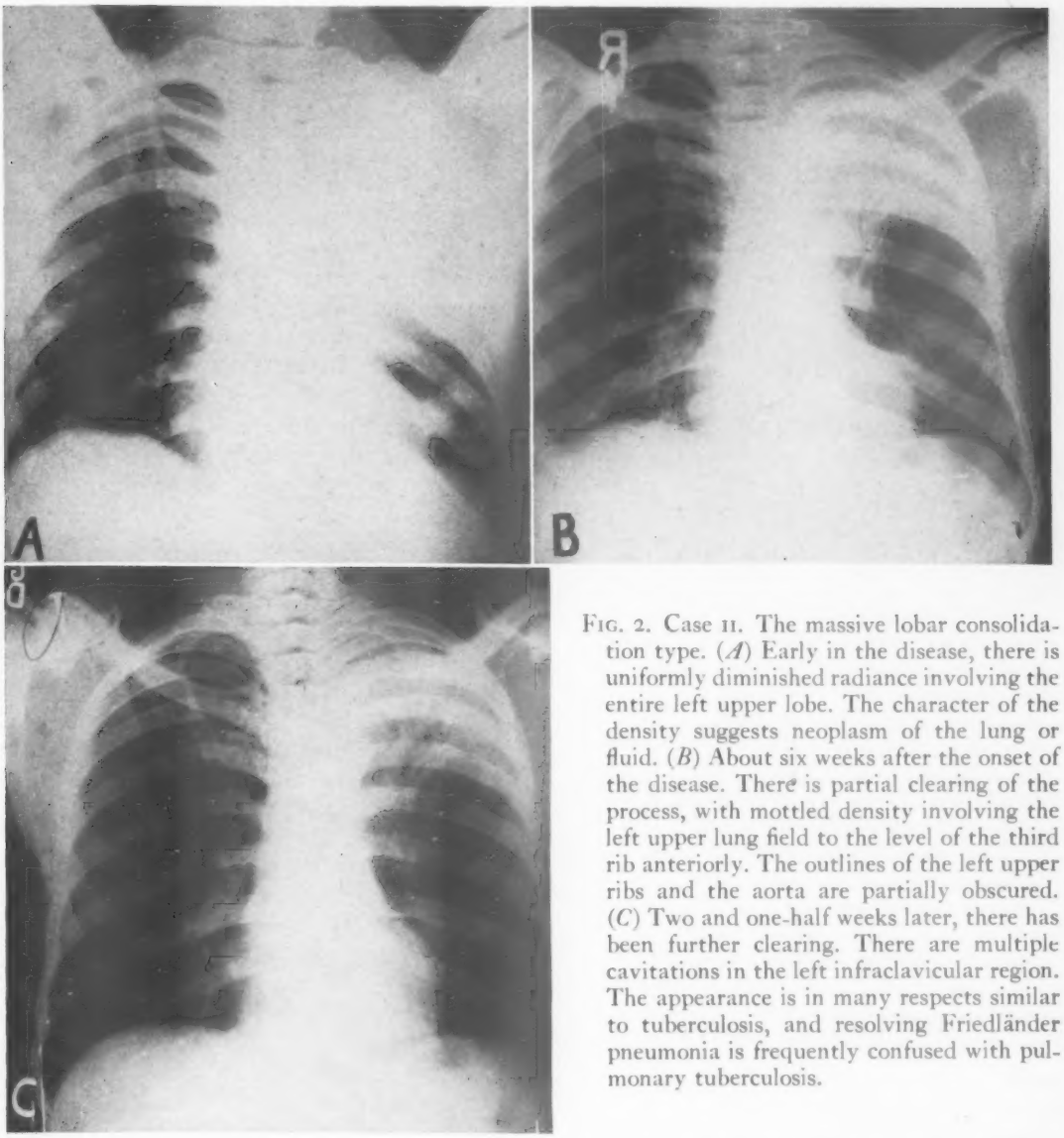

Fig. 2. Case II. The massive lobar consolidation type. (*A*) Early in the disease, there is uniformly diminished radiance involving the entire left upper lobe. The character of the density suggests neoplasm of the lung or fluid. (*B*) About six weeks after the onset of the disease. There is partial clearing of the process, with mottled density involving the left upper lung field to the level of the third rib anteriorly. The outlines of the left upper ribs and the aorta are partially obscured. (*C*) Two and one-half weeks later, there has been further clearing. There are multiple cavitations in the left infraclavicular region. The appearance is in many respects similar to tuberculosis, and resolving Friedländer pneumonia is frequently confused with pulmonary tuberculosis.

tuberculosis. He was obviously very acutely ill and disoriented. Temperature 103°F., pulse 116, respirations 50.

Laboratory data: White blood cell count 12,400 (polymorphonuclears 68 per cent) on admission. Two days later the white blood cell count was 12,000. The sputum culture showed *Bacillus mucosus capsulatus* Group A and when repeated on April 9 and May 5 the same organism was present. Blood cultures developed no growth.

Course. The patient was given oxygen, penicillin, and sulfadiazine. After a stormy course during forty-eight hours he improved rapidly, the temperature dropping to 98.6° F. within four days. Roentgen studies did not demonstrate corresponding clearing of the pulmonary condition, however. Fluid with cavitation in the left upper lobe developed subsequently. Tuberculosis was suspected but all tests to demonstrate the organism were unsuccessful and guinea pig inoculation was negative. After being ambulatory and asymptomatic for a month the patient was discharged from the hospital although the roentgenogram had not cleared completely. He has been observed in the Out-Patient Department and has continued well.

Case III. The lobular consolidation type (Group 2). J. S., male, aged forty-four, laborer,

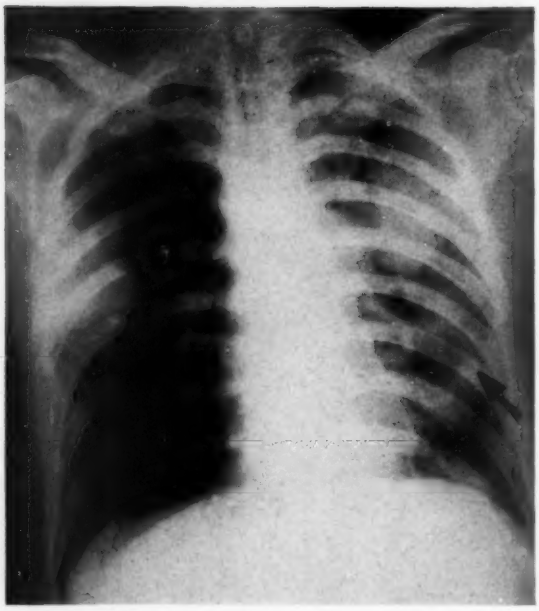

Fig. 3. Case III. Friedländer pneumonia, lobular type. Roentgenogram on the day of admission to the hospital reveals irregular, mottled density throughout the entire left lung field and in the axillary portion of the right upper and mid-lung field. There is a rounded area of radiance measuring about 4 cm. in diameter in the left lung field slightly below the angle of the scapula (arrow) in the region of the seventh and eighth interspaces, consistent with a cavitation.

admitted December 7, 1944, with a history of not having felt well for the past three weeks. Tiredness, cough with yellow sputum, and occasional chills were the first complaints. Subsequently he developed a sore throat, pain in the chest on deep inspiration, coughing, general malaise, and increasing dyspnea. There was a long history of chronic alcoholism. On physical examination, he was described as "poorly nourished, dyspneic, with foul odor to breath." Decreased expansion, dullness, coarse and fine, moist rales were present throughout the left lung. The breath sounds and vocal fremitus were decreased. There was a friction rub in the anterior axillary line in the fifth and sixth interspaces.

Laboratory data: Blood pressure 110/60, temperature 102° F., pulse 110, respirations 42. Urine, negative. Red blood cell count 3.54 million, leukocyte count 22,300 (normal differential count). Sputum culture revealed *Bacillus mucosus capsulatus* on two occasions. The blood culture showed no growth. Roentgen examination of the chest demonstrated lobular pneumonia involving the left upper lobe, the left lower lobe, and the right upper lobe with an abscess formation in the left inferior lung field.

Course. The patient appeared very ill on admission. He was placed on sulfadiazine, oxygen, and supportive measures. The white blood cell count, which on admission was 22,300, decreased to 8,500 on the sixth hospital day. The temperature remained elevated and dyspnea was a constant feature. He grew progressively worse and died on the ninth day.

Case IV. The chronic form characterized by abscess formation and chronic suppuration (Group 3). M. C., male, aged fifty, entered the hospital on November 7, 1946, because of sudden onset of severe left chest pain and a chill. Cough, nausea, and vomiting occurred several times before admission. There was a history of three previous admissions with recurrent Friedländer pneumonia. On physical examination, there was splinting of the left side of the chest with decreased breath sounds and dullness over the left lower chest. A friction rub and coarse dry rales and rhonchi were present.

Laboratory data: White blood cell count 15,600 (79 per cent polymorphonuclears, 2 per cent monocytes). Sputum cultures showed *Bacillus mucosus capsulatus* Type A and were negative for the tubercle bacillus. Roentgen examination demonstrated mottled density at the left base and linear strands of fibrosis overlying the right diaphragm.

Course. During his stay in the hospital a left maxillary sinusitis and ethmoiditis was found. *Bacillus mucosus capsulatus* was isolated from cultures of the left antrum. The temperature on admission was 99° F. and rarely rose above normal. Although the patient improved clinically the organism persisted in the discharge from the sinuses and in the sputum. Streptomycin was administered with the aerosol apparatus for ten days. Cultures taken from the nose and throat became negative for Friedländer's bacillus after the second day of treatment and remained so until he was discharged well on January 21, 1946.

Case V. The chronic form characterized by abscess formation and suppuration (Group 3). J. W., male, white, aged fifty-nine, was admitted September 21, 1946, with a history of a

"cold for the past week." Two days previously he had developed severe pleuritic pain in the right anterior chest and a cough productive of purulent sputum. On admission, he appeared acutely ill and in a shock-like state. The skin was flushed, tongue dry, lips and finger nails cyanotic. Temperature 98.6° F., pulse 120, respirations 32, blood pressure 95/55. There were signs of consolidation over the lower half of the right chest anteriorly and a few medium crepitant rales throughout the left chest posteriorly. The heart was not enlarged and no murmurs were heard; the rhythm was irregular.

Laboratory data: Hemoglobin 110 per cent, leukocyte count 25,000 (96 per cent polymorphonuclears with 50 per cent young forms). The sputum was tenacious, purulent, and bloody. A stained smear showed innumerable, large, encapsulated, gram-negative bacilli and a few gram-positive diplococci. A Neufeld test with antipneumococcus serums showed "quellung" of some of the diplococci with Type 19 antiserums. Cultures of the sputum on blood agar yielded only Friedländer's bacilli and this organism and Type 7 pneumococci were obtained from cultures of the heart's blood of a mouse inoculated with the sputum. The blood culture on admission was positive for Type C Friedländer's bacillus.

Course. Streptomycin was administered intramuscularly 1 gram every four hours for two days and then at six hour intervals for four days. The patient's general condition began to improve within a few hours. He became stronger and his appetite returned. The pleuritic pain diminished and the dyspnea lessened. Subsequent blood cultures were negative.

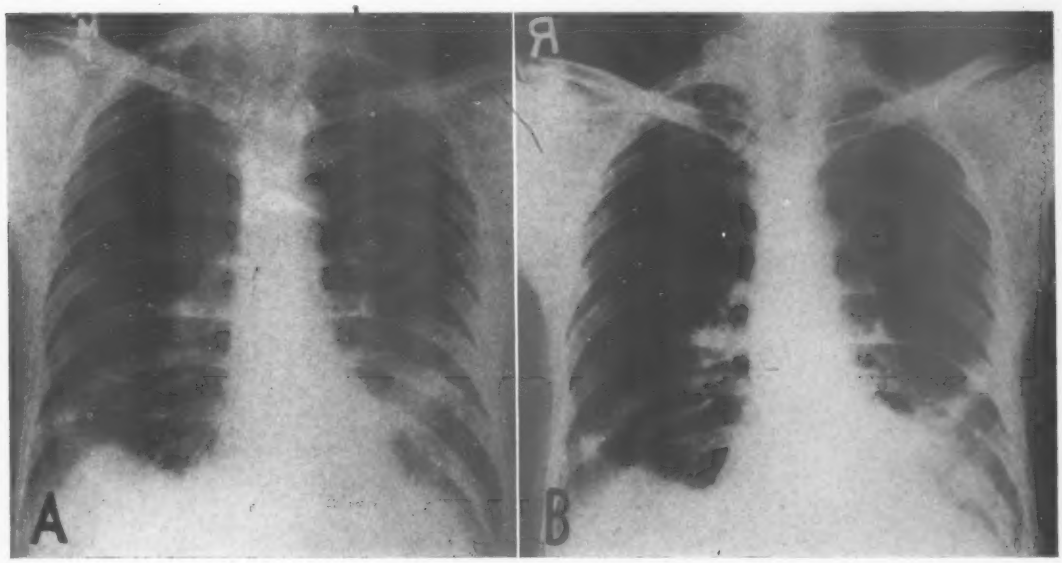

Fig. 4. Case IV. The chronic type with abscess formation and suppuration. (*A*) Thirteen days after onset. There is a diffuse, irregular mottling involving the lower third of the left lung field and the right base. There are multiple small cavitations on the left. (*B*) About six weeks later. The process is less marked, indicating moderate clearing. There are small cavities at the left base. The right diaphragm is elevated and irregular and the costophrenic angle is shallow and hazy.

Friedländer's bacilli persisted in the sputum in varying numbers. Type 7 pneumococci were isolated on one occasion and Type 19 were found several times. On the fifth day, signs of cavitation of the right middle lobe were elicited clinically and confirmed by the roentgenogram. Streptomycin was discontinued intramuscularly and subsequently was given by inhalation of 125 mg. dissolved in 1 ml. of saline ten times daily. Each inhalation was preceded by a five minute period of postural drainage. The patient improved and the number of Friedländer's bacilli in the sputum decreased. On the tenth day, because of the persistence of large numbers of Type 19 pneumococci in the sputum and elevation of the temperature, the patient was again given intramuscular penicillin, 100,000

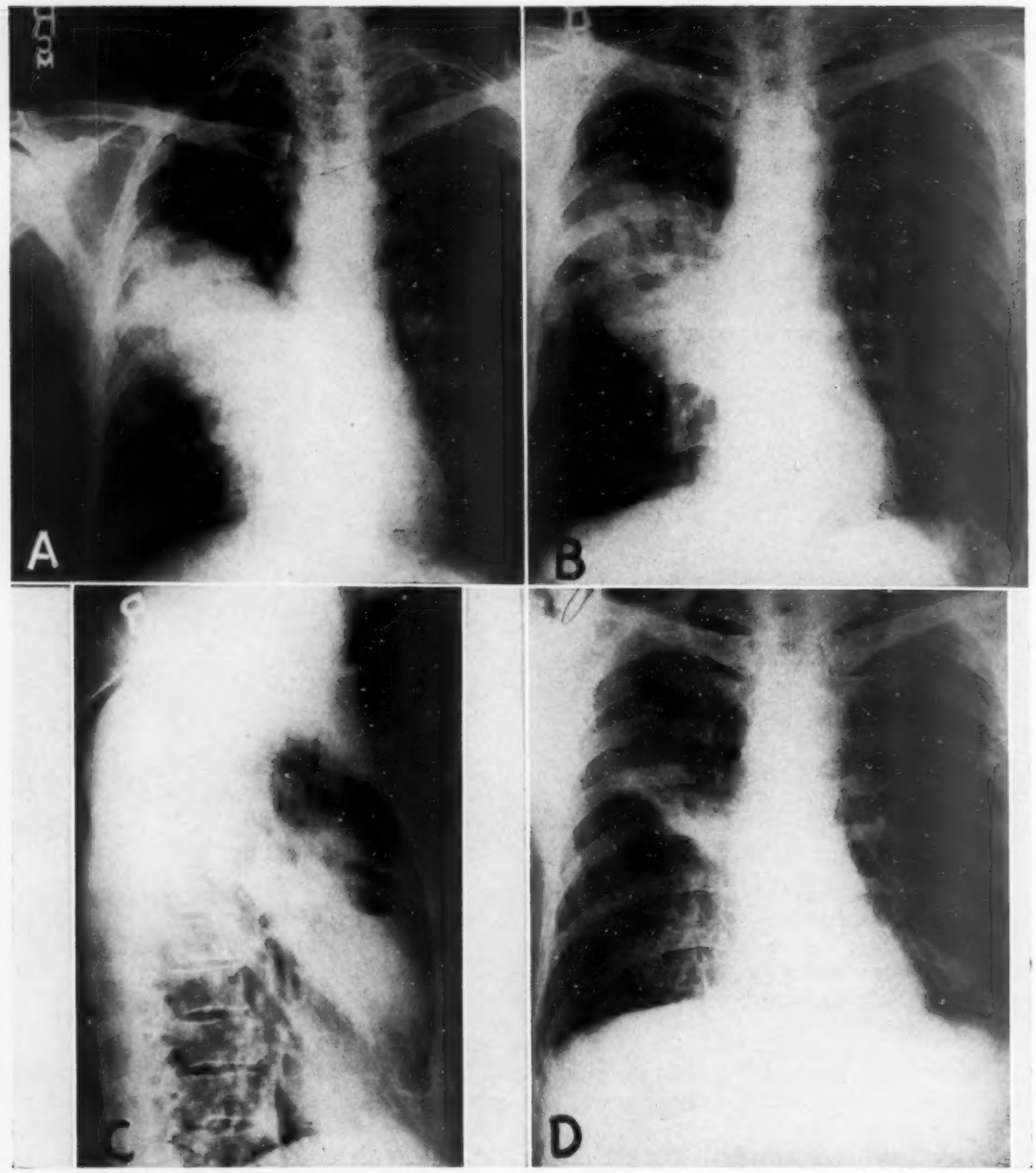

Fig. 5. Case v. The chronic type with abscess formation. (*A*) Two days after onset. There is an area of marked density in the right mid-lung field. The margins of the affected area are sharply defined. The left lung is unaffected. (*B*) Eleven days later. There is a large cavitation within the involved area. (*C*) Same day as in (*B*), lateral projection, patient standing. The fluid level in the cavity is clearly demonstrable. (*D*) Seven weeks after the onset of the illness. The process has cleared markedly and there is now a narrow band of density adjacent to the right hilum.

units every eight hours. The temperature dropped promptly and the number of pneumococci in the sputum diminished. Both penicillin and streptomycin were discontinued on the seventeenth day. At this time the patient had only a slight residual cough, raised moderate amounts of purulent sputum, and had some sweating at night. The roentgenograms still showed an area of consolidation with several small abscesses. During the following two months the patient remained afebrile and symptom free and regained much weight and

strength. The area of pulmonary consolidation diminished in size and showed evidences of fibrosis. The abscesses became smaller and finally disappeared completely. Friedländer's bacillus continued to be the predominant organism in the sputum cultures, Type 19 pneumococci not being found after the third week.

SUMMARY AND CONCLUSIONS

Friedländer pneumonia is relatively infrequent. Nevertheless it is a very important type of pneumonia because of its severity, the high incidence of complications, and the markedly elevated mortality rate. Early recognition of the disease and the prompt institution of therapy are of the utmost importance. The clinical manifestations are described.

The roentgen findings are variable. There is a massive lobar consolidation variety, a lobular form, and a type characterized by chronicity with lung abscess formation and pulmonary suppuration. It is in the massive lobar consolidation variety of the disease that the roentgenologist may be of the greatest aid to the clinician, although the roentgen studies are also of value in the demonstration of the other types of Friedländer pneumonia.

Streptomycin appears beneficial in some cases of Friedländer pneumonia especially if commenced early in the acute phase. Penicillin and the sulfonamides do not appear to have influenced the mortality in this disease to any appreciable extent.

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A CASE OF ANEURYSM OF THE DUCTUS ARTERIOSUS WITH POSTMORTEM ROENTGENOLOGIC STUDY AFTER INSTILLATION OF BARIUM PASTE

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WHEREAS simple patency of the ductus arteriosus is second only to patent foramen ovale as the most common congenital lesion of the heart, aneurysm of the ductus arteriosus is indeed rare. Mackler and Graham[13] reviewed the literature in 1943 and concluded that only 30 cases of true ductus aneurysm had been reported, including the one reported by them at that time. Because of the rarity of the condition, and because of its interest to the roentgenologist and the thoracic surgeon as one type of mediastinal tumor, the author reports the following case of aneurysm of the ductus arteriosus found at the postmortem examination of a three weeks old infant.

HISTORICAL

The first cases of aneurysm of the ductus arteriosus were described by French authors with Billard[1] in 1837, Martin St. Ange and M. Parise,[16] Geoffrey St. Hilaire and Baron[17] reporting a total of 4 cases. Thore[20] in 1850 reviewed the cases previously reported by the French and added 8 of his own. Other reports of aneurysm of the ductus arteriosus which are now generally regarded as being cases of true ductus aneurysms include one reported by Hebb[10] in 1893, three cases described by Roeder[15] in 1901, two by Esser[3] in 1902, one case by Grunner[7] in 1904, and a case reported by Thorel[21] in 1904. Subsequent cases were reported by Weller[22] in 1915, Frolicher[4] in 1917, one by Schattman[18] in 1919, and one case reported by Dry[2] in 1921. Hammerschlag[9] reviewed the literature and added a case of his own in 1925. Guggenheim[8] in 1930 and Scheef[19] in 1939 each reported cases of true aneurysm of the ductus arteriosus. Graham[5] reported 2 cases of ductus aneurysm in 1940. Mackler and Graham[13] reported another case in 1943 and reviewed the literature thoroughly at that time.

ETIOLOGY OF ANEURYSM OF THE DUCTUS ARTERIOSUS

The etiology of aneurysm of the ductus arteriosus is not clear. Langer, Walkhoff and Thoma,[12] Kautsch and Klotz[11] as cited by Dry,[2] all pointed out that the ductus arteriosus is normally deficient in elastic tissue but is relatively rich in smooth muscle fibers. Dry concluded from his own case of aneurysm of the ductus arteriosus that the probable cause was a deficient development in both the elastic and the muscular elements of the ductus arteriosus. In Dry's case practically no elastic tissue and no intimal lining layer were found in the microscopic study of the specimen. Gross[6] assumed that a thin or a structurally deficient wall of the ductus arteriosus was the main predisposing factor in its aneurysmal dilatation. Esser[3] was able to demonstrate dissecting hemorrhages in the media of the ductus in his 2 cases. Frolicher[4] also found hemorrhages in the media in his case. Mackler and Graham[13] reviewed the literature and concluded that infectious processes could only be ascribed to be the etiologic factors in 3 of the 30 reported aneurysms. These authors advanced the theory that the basis for aneurysm of the ductus arteriosus lay in the inherent characteristics of that structure itself. They believe that the normal lack of elastic tissue associated with the abundance of smooth muscle is the natural predisposing factor to dilatation of a patent ductus arteriosus, when the latter is subjected to the high pressure of the blood within the aorta.

Ductus aneurysms are rarely found in adults. Of the 30 reported cases only 6 were present in adults, with the average age being thirty-one years. All others were

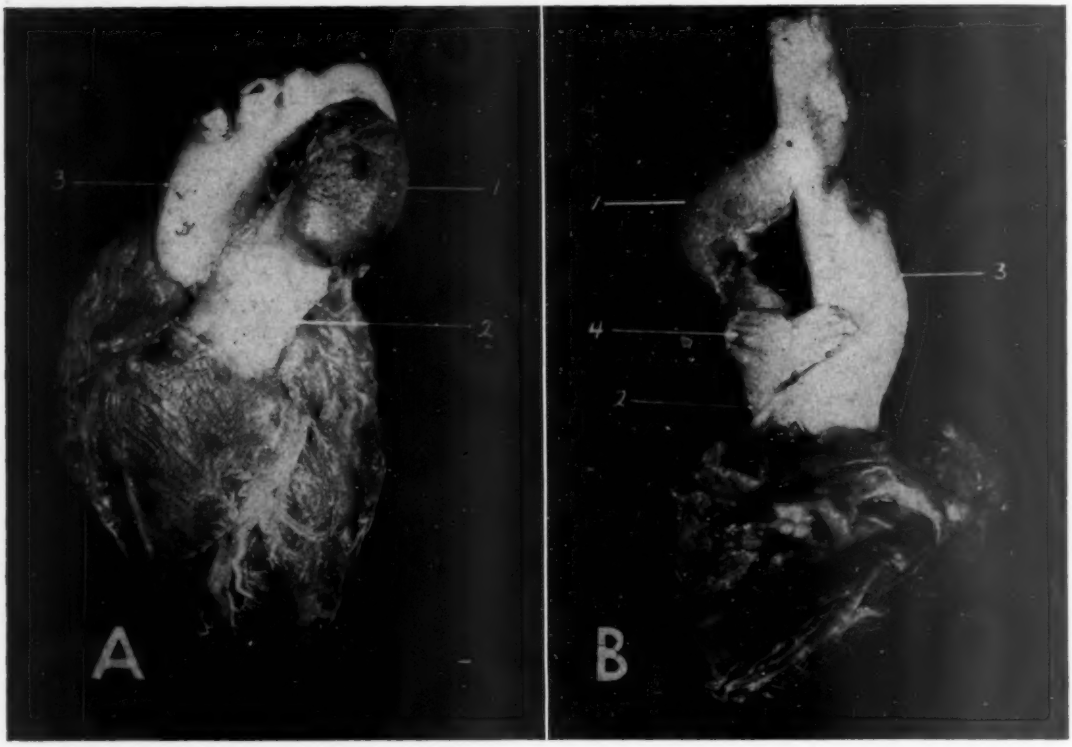

Fig. 1. Anterior view (*A*) and posterior view (*B*). (*1*) aneurysm of the ductus arteriosus, (*2*) pulmonary artery, (*3*) aorta, and (*4*) the left pulmonary artery. These views show the ductus aneurysm to extend anteriorly and to the left as an elliptical rounded structure. The pulmonary ostium of the aneurysm is proximal to the bifurcation of the pulmonary artery. Observe the dark color of the aneurysm as compared to the whitish appearance of the aorta and the pulmonary artery. This darker color is probably due to the lack of fibrous elastic tissue and the abundance of smooth muscle in the aneurysm. (These photographs are approximately 2× the actual size of the specimen.)

found during the postmortem examination of infants ranging from a few days to two months of age.

CASE REPORT

The patient was a premature female Negro infant of eight months gestation, born on October 8, 1947, and brought to the Indianapolis General Hospital because of prematurity. Birth weight was 3 pounds and 12 ounces. There were no unusual physical findings other than the prematurity of the infant.

The course in the hospital was uneventful for the first three weeks. However, at 6:00 P.M. on October 29 the infant's condition suddenly took a turn for the worse. For no apparent reason the infant passed into a shock-like state. The eyes, arms, and legs were noted to twitch repeatedly and appeared spastic. Breathing was of the gasping type, and even oxygen failed to relieve the dyspnea. The skin became cold and clammy to the touch. The temperature fell to 96° F. Death occurred the following morning at 3:00 A.M.

Autopsy Report. The body was that of a premature Negro female infant measuring 40 cm. over-all length. The face and the upper portion of the body appeared cyanotic. No distinguishing external features were seen other than those of a general debility and poorly nourished state.

The interesting features of the case were found in the thoracic and cranial cavities. Examination of the thoracic cavity revealed a tumor of firm consistency located between the pulmonary artery and the aorta in the region normally occupied by the ductus arteriosus. This tumor measured 18 mm. in length and 14 mm. in width. It was cylindrical in shape but bulged anteriorly. Further examination and palpation revealed the tumor to be filled with clotted blood. With probing it was then obvious

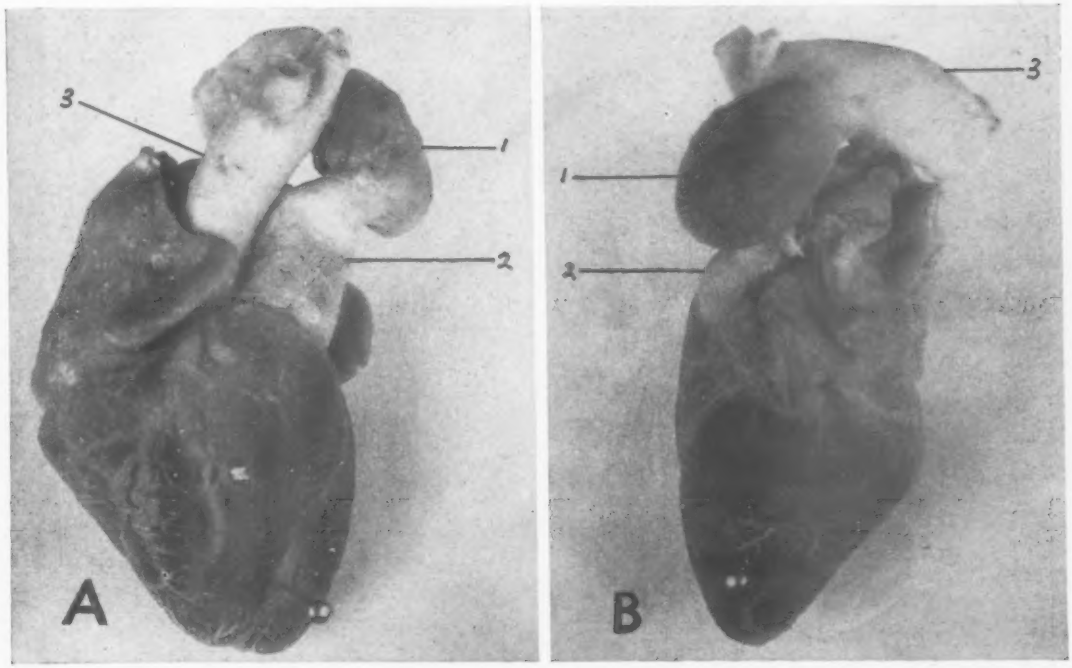

FIG. 2. Right anterior oblique view (*A*) and left lateral view (*B*). (*1*) ductus aneurysm, (*2*) pulmonary artery, and (*3*) aorta. The aneurysm of the ductus is seen to extend downwards, anteriorly and to the left in these views. (These photographs are approximately 2.5 × the size of the specimen.)

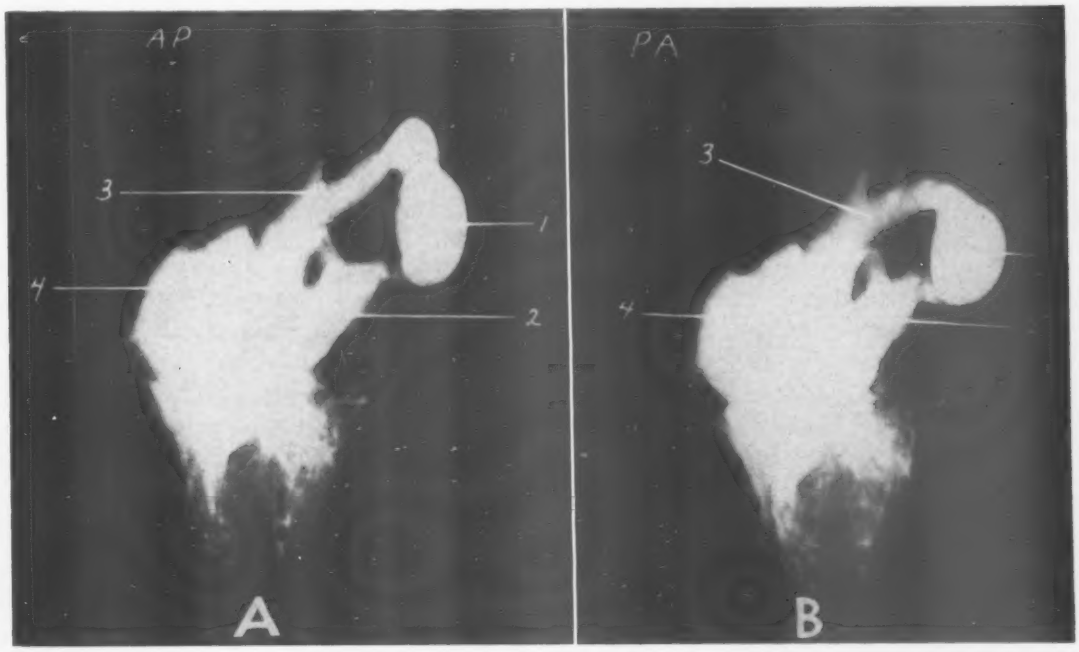

FIG. 3. Roentgenograms following the injection of barium paste. Anteroposterior view (*A*) and postero-anterior view (*B*). (*1*) ductus aneurysm, (*2*) pulmonary artery, (*3*) aorta, and (*4*) right ventricle. The aortic and pulmonary ostia are visualized at the upper and lower aspects of the aneurysm respectively. The aneurysm presents itself as a rounded elliptical shadow well to the left of the cardiac silhouette, and roughly located in a position between the aortic arch and the pulmonary conus.

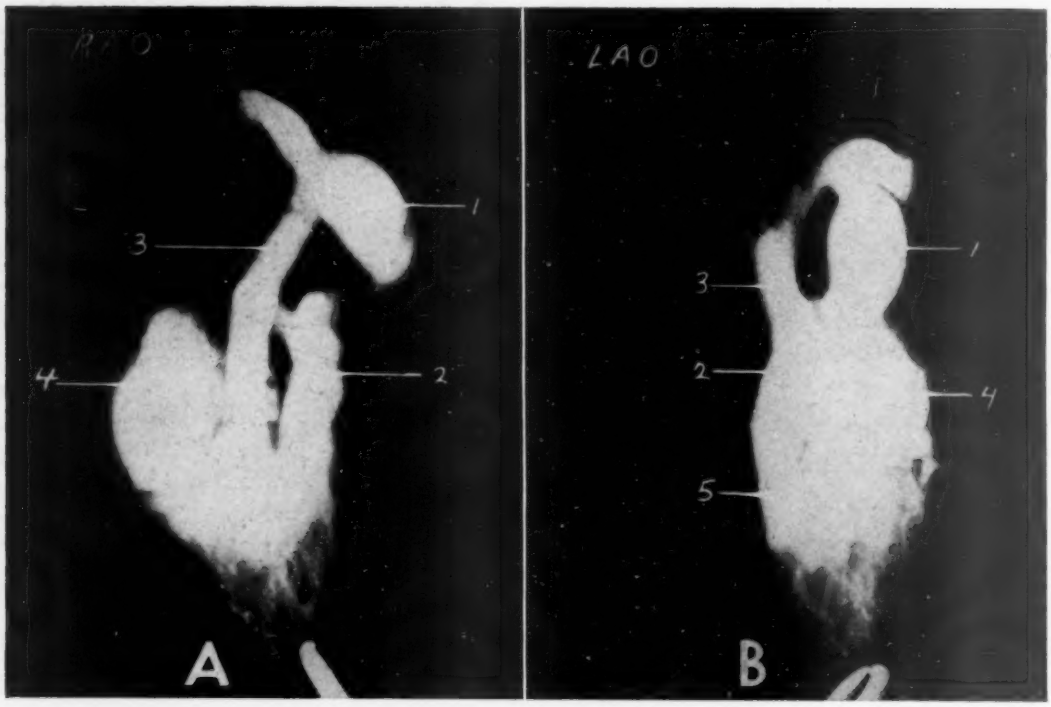

Fig. 4. Right anterior oblique view (*A*) and left anterior oblique view (*B*). (*1*) ductus aneurysm, (*2*) pulmonary artery, (*3*) aorta, (*4*) right auricle, and (*5*) right ventricle. In the right anterior oblique view the ductus aneurysm forms a rounded shadow above and to the left of the pulmonary conus. The aneurysm lies between the bulge produced by the conus and that of the aortic arch. In the left anterior oblique view the ductus aneurysm is seen to be posterior to the ascending limb of the aorta and within the area bounded anteriorly by the ascending aorta, superiorly by the aortic arch and posteriorly by the descending aorta. The so-called "aortic window," normally seen in this view, is essentially obliterated by an aneurysm of the ductus arteriosus.

that the tumor was an aneurysm of the ductus arteriosus.

On opening this ductus aneurysm, the wall was seen to be very thin and friable and one portion was discolored and appeared necrotic. From the gross examination the aneurysm appeared to be near rupture. The ostium at the aortic end measured 4 mm. in width and the pulmonary opening measured 2 mm. Microscopic study of the wall of the aneurysm revealed little if any elastic tissue but a large amount of smooth muscle.

Examination of the brain revealed a softening of the cerebral region normally supplied by the right internal carotid artery. This was believed to be due to a cerebral infarction secondary to an embolus which had its orign in the ductus aneurysm and which had lodged in the right internal carotid artery. Through an oversight the right internal carotid artery was not completely examined. Microscopic study revealed a typical early infarction of the right cerebral hemisphere.

Microscopic study of the lungs, liver, spleen, and the kidneys revealed a generalized congestion.

Pathologic Diagnosis: (1) aneurysm of the ductus arteriosus; (2) right cerebral infarction, secondary to (1); (3) congestion of the viscera.

DISCUSSION

The main value of this paper is in the accompanying illustrations of the pathologic specimen and the reproductions of the roentgenograms which were taken after the heart had been injected with a thick barium paste. Unfortunately no roentgenograms during life were taken, but it is only reasonable to assume that the features of the ductus aneurysm described with the illustrations would be the same or very similar to those produced during life. There is no doubt that the ductus would fill with contrast media such as diodrast during life,

using the method of Robb and Steinberg.[14] Such filling would occur from the aorta because of the higher pressure within that vessel as compared to that within the pulmonary artery. The findings during life should be essentially the same as those shown in the accompanying illustrations of roentgenograms.

There are only 4 cases reported in which roentgen studies of ductus aneurysms were made during life, i.e., Scheef's case, Graham's 2 cases, and the case of Mackler and Graham in which the correct preoperative diagnosis was made from the roentgen study. Scheef had a chest roentgenogram (posteroanterior view) which showed the ductus aneurysm to produce a smooth rounded shadow of increased density in the left hilar region. Mackler and Graham pointed out that the most important feature in the roentgen diagnosis of ductus aneurysm was the peculiar location of the tumor. In all 3 of their cases they found the aneurysm to show as a tumor in the superior mediastinum, in the region of the pulmonary conus and lying posterior to the trachea. In 1 of their cases the periphery of the aneurysm showed as a distinct shell of calcification. In all 3 cases the trachea was displaced anteriorly and to the right by the tumor. Evidence of pulsation was sought for in all 3 of their cases and was found to be of the expansile type in only one. Graham explains this discrepancy on the basis of a laminated clot within the aneurysm and stresses the point that absence of pulsation should not keep one from making the diagnosis of ductus aneurysm if the other points are in favor.

From the case reported here it is obvious that the aneurysm has not enlarged or extended posteriorly, but instead has gone anterior to the trachea and the left bronchus. This is the only feature of the present case which is not in accordance with the diagnostic criteria laid down by Mackler and Graham in whose cases the aneurysm was always posterior to the trachea, displacing that structure anteriorly and to the right. Possibly this anterior extension of the ductus aneurysm in the present case was due to the origin of the ductus arteriosus from the pulmonary artery proper rather than from the left pulmonary artery. At any rate it cannot be considered as a hard and fast rule that all aneurysms of the ductus arteriosus lie posterior to the trachea for they may be anterior to it as in the case reported here.

Fig. 5. Left lateral view. (*1*) ductus aneurysm, (*2*) pulmonary artery, (*3*) aorta, (*4*) right auricle, and (*5*) right ventricle. In the lateral view the ductus aneurysm is seen to extend anteriorly, producing a prominent bulge anterior to both the pulmonary conus and the aorta and again located roughly between these two landmarks. Such an aneurysm would present itself as a tumor of the anterior mediastinum and in the lower portion of the superior mediastinum. This tumor would then be anterior to the trachea and the left main stem bronchus.

SUMMARY

(1) Aneurysm of the ductus arteriosus is a rare condition with thirty-one cases now reported. However, it is of importance to the roentgenologist and the thoracic surgeon as one type of mediastinal tumor. The attempted removal of such a tumor could result in uncontrollable hemorrhage and it would be well for a thoracic surgeon to be aware of the potentialities of such a complication.

(2) The peculiar location of such a tumor is its most significant feature:

(a) In posteroanterior and anteroposterior views the aneurysm of the ductus arteriosus extends to the left and slightly above the pulmonary conus.

(b) In the right anterior oblique view the aneurysm is projected as a tumor mass to the left and superior to the pulmonary conus and between the latter and the aortic arch.

(c) In the left anterior oblique view the clear shadow of the "aortic window" is obliterated by the tumor produced by the ductus aneurysm.

(d) The lateral view in the present case shows the aneurysm to be anterior to the trachea and to extend farther anteriorly than either the pulmonary conus or the aorta. This is the only feature of the present case not in accordance with the diagnostic criteria laid down by Mackler and Graham who found that the aneurysm was posterior to the trachea in their three cases.

(e) The ductus aneurysm would produce a tumor shadow in the anterior mediastinum and in the lower portion of the superior mediastinum.

(3) Visualization of the aneurysm with diodrast using the technique of Robb and Steinberg should establish the diagnosis.

I wish to acknowledge the help I received from the Departments of Pathology and Radiology at the Indianapolis General Hospital and especially the kind assistance of Dr. W. B. Dublin.

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COCCIDIOIDAL GRANULOMA OF BONE

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COCCIDIOIDOMYCOSIS is produced by a specific fungus, *Oidium coccidioides* or *Coccidioides immitis*, as first suggested in 1896 by Rixford and Gilchrist.[5] Ophüls and Moffit[6] reported a case in 1900 and were the first to demonstrate the organism to be a mold. The infectious form in animals, parasitic phase, is a spherule with a double refractile wall. These may vary in size from 10 to 200 microns in diameter. Within the spherule numerous endospores are formed which are released to spread within the animal host by way of the blood stream or lymphatics when the wall of the spherule ruptures. The saprophytic phase consists of long septate hyphae producing a mycelium.

The disease process is endemic in Southern California in the Central Valley and the Mojave Desert. Cases have been reported from Arizona, as well as from various parts of the country. During World War II, many thousands of troops were stationed or underwent training in endemic areas of California and were exposed to possible infection by the coccidioidomycosis. Hundreds of migrant agricultural workers, drawn from the racial groups which are the most susceptible to the disease process, are exposed to infection every year in Kern County.

In 1931, Carter[2,3] reported on 50 cases of coccidioidal granuloma in bone, among whom there were 94 lesions. He described in detail the sites of involvement. In 1934, he[3] again reported on the cases which he had seen up to that time, now amounting to 70.

Benninghoven and E.R. Miller[1] reviewed the cases up to December, 1941, at Kern General Hospital, Bakersfield, and included them in their review along with cases from Los Angeles County Hospital and Stanford Hospital.

We have collected 17 cases (Table 1) from July, 1942, to October, 1947, illustrating bone lesions of coccidioidal granuloma and which were confirmed clinically by skin tests, serological study, biopsy, culture or on sections taken at autopsy.

The cases were all positive for either one or more of the above tests.

As can be seen from Table 1, there were 8 Negroes, 4 Filipinos, 3 white persons, 1 Mexican, and 1 Chinese. This is consistent with the findings of other observers that the most susceptible individuals are members of the colored races.

The duration of symptoms before the patient presented himself with complaints involving the osseous system varied from thirty days (Case 7) to sixteen years (Case 2).

At the time the examinations of the bone lesions were made there was lung involvement in 9 of the cases. Pulmonary involvement was not present in 8 of the cases.

Table II shows the distribution of lesions among bones.

Case 7 (Table 1), a Filipino, aged thirty-four, developed a painless, growing mass over the right sternoclavicular joint thirty days before presenting himself for treatment. On roentgen examination, there was involvement of the medial end of the clavicle.

All bone lesions showed marked variations similar to those described by Benninghoven and Miller,[1] and Carter.[2,3] However, in our series, there was no bone involvement caused by extension from a soft tissue abscess or granuloma in the region of the bony structure. This is contrary to the findings of Carter and Benninghoven and Miller who stated "that there was invasion of underlying bony structure by extension from an overlying abscess". On the other hand, in our series of bone cases, the bone lesions may cause the soft tissue involvement which will go on to ulceration and

TABLE I

Case	Race	Age	Duration	Lung Involvement	Bone Lesions	Skin Test	Serological Findings	Other Laboratory Procedures
1—A.W. Male	Colored	24 yr.	7 mo.	Positive	Single rib, 5th left side, ant.	Negative	Positive on 2-13-47 and 3-19-47 with increasing titer	
2—W.H. Male	Colored	21 yr.	16 yr.	Negative	Lumbar spine, ant.	Positive	Positive	
3—B.A. Male	Colored	15 yr.	22 mo.	Negative	2nd metacarpal 5th metatarsal	Negative	Positive	Sequestrectomy 2nd metacarpal
4—I.A. Male	Filipino	38 yr.	6 wk.	T.B. neg. cocc.	Navicular, patella left leg; femur left leg	No record	No record	Surgical removal of navicular; showed microscopic positive for coccidioidomycosis
5—W.V. Male	White	42 yr.	4 yr.	Positive	Talus, navicular left foot; patella right knee; lat. condyle rt. elbow	Positive	Positive	
6—L.C. Female	Colored	9 yr.	7 yr.	Negative	Multiple bone involvement, especially T_{12} and L_1	Positive	No record	
7—B.U. Male	Filipino	34 yr.	30 days	Positive	Rt. sternoclavicular joint; medial end clavicle	Negative	No record	Aspiration biopsy of mass of clavicle with positive culture
8—F.L.N. Male	White	33 yr.	5 yr.	Positive	Left ilium	Positive	Positive	Positive culture
9—M.A.M. Female	White	69 yr.	2 mo.	Positive	Skull, left tibia	Negative	Died	Sections of lesions positive for coccidioidal granuloma of bone
10—E.N. Female 6 mo. pregnant	Colored	37 yr.	3 mo.	Positive	T_4; T_5; L_2	Negative	Positive	Biopsy of ilium positive; autopsy specimen
11—C.B. Female	Colored	37 yr.	4 wk.	Positive	L_4; S_1; S_2; sternum	Negative; Mantoux positive	No record	Autopsy
12—G.W. Male	Colored	38 yr.	2½ mo.	?	Distal end radius	Positive	Positive	Culture positive
13—M.L. Female	Chinese	65 yr.	6 mo.	Positive	Both knees, patellae, tibiae and femur	No record	No record	Culture positive
14—A.B. Male	Filipino	41 yr.	2 mo.	Positive	L_4, L_5	Positive	Positive	Culture positive
15—J.S. Male	Filipino	30 yr.	4 mo.	Positive	Cuboid; distal third of clavicle	Positive	Positive	Positive on examination of smear
16—R.E. Male	Mexican	26 yr.	1 yr.	Positive	Lumbar spine L_1 to L_4	No record	Positive	Autopsy
17—J.S. Female	Colored	26 yr.	4 mo.	?	Sternum	Negative	Positive	Autopsy; microscopic sections positive

subsequent abscess formation.

In 6 cases the skin test was negative in dilutions as low as 1 to 100 for coccidioides, while the serological test was positive in every case in which the serological examination was done. All serological tests were performed by Dr. C. E. Smith, of Stanford Medical School. In our series of cases, a negative coccidioidin skin test may be of no value in ruling out the disease process. Serological tests, cultures and biposies are

TABLE II

DISTRIBUTION OF LESIONS AMONG BONES

Skull	1	Tarsal navicular	1
Clavicle	2	Cuboid	1
Radius	1		
Metacarpal, 2nd	1	Vertebral column:	
Metatarsal, 5th	1	T-6	1
Sternum	2	T-8	1
Ribs	1	T-12	1
Ilium	2	L-1	1
Femur	4	L-2	2
Patella	3	L-3	3
Tibia	3	L-4	3
Fibula	1	S-2	1

TABLE III

Age Groups	No. of Cases
1–10	1
11–20	1
21–30	4
31–40	7
41–50	2
51–60	0
61–70	2

invaluable aids in establishing the diagnosis of coccidioidomycosis.

The age distribution shows that the majority of cases of disseminated coccidioidomycosis occur mostly in the third and fourth decades; that is, when an individual's activity is greatest (Table III).

From Table I we have selected certain cases for illustration.

REPORT OF CASES

Case 1. A. W. This patient was first seen in January, 1947, with chief complaints of pain between the shoulder blades of about four days' duration. Previous to this he had had a slight cold which had been present for about five weeks. Pain in the back was much worse on breathing, was fairly steady and did not radiate. The patient had a slight cough and temperature of 101° F. There was no hemoptysis. There had been about a 15 pound weight loss in the month previous to admission.

Physical findings revealed dullness over both lower lung fields, diminished breath sounds, especially on the left, and bronchial breathing in the right lower lung field. Roentgen examination at this time showed some infiltration in both lower lung fields. Figure 1 shows a roentgen study of the chest taken on February 11, 1947. There were a few linear densities in the right base; however, there was a marked infiltration in the left lower lung field which had the appearance of a consolidation.

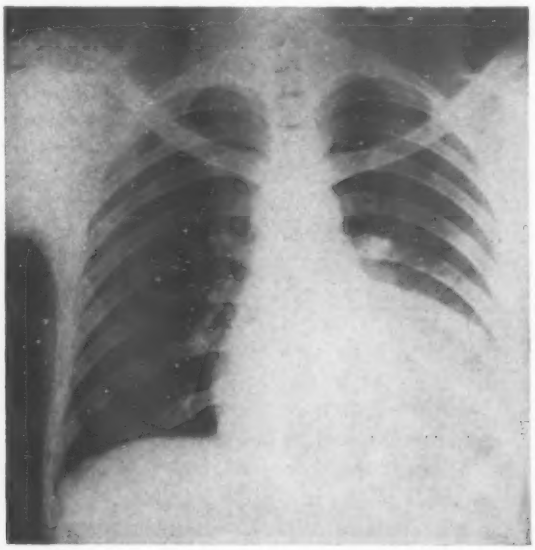

Fig. 1. Case 1. Shows pneumonic infiltration, left lower lung field, extending down from left hilar area to involve lower half of left lung.

Fig. 2. Case 1. Shows a clearing of infiltration in left lower lung field but with beginning erosion of the fifth rib along the superior margin anteriorly.

The patient developed nodules in the antecubital fossa of the right arm, and in the middle third of the right leg. On February 19, 1947 biopsies were taken. The microscopic report showed an inflammatory granuloma. Serological tests for coccidioidal infection, done on February 21, 1947, revealed a complement fixation which was positive for coccidioides. There was subjective improvement during the patient's stay in the hospital. Pain in the left leg gradually disappeared. A follow-up visit in the Chest Clinic on April 10, 1947, and a roentgen study of the chest at that time, showed that the inflammatory process in the left lower lung had almost disappeared. In Figure 2 it can be noted that there was beginning erosion of the fifth rib on the left side, anteriorly.

Toward the end of April, a lump on the anterior left lower chest was discovered which was a little larger than the size of an egg. At about this same time, swelling of the right thigh developed. On May 6, 1947, further roentgen studies of the chest revealed an increse in the erosion of the fifth rib. Soon after this, the lump in the anterior left lower chest went on to abscess formation and suppuration, and began to drain. Marked erosion of the fifth rib, left side, was

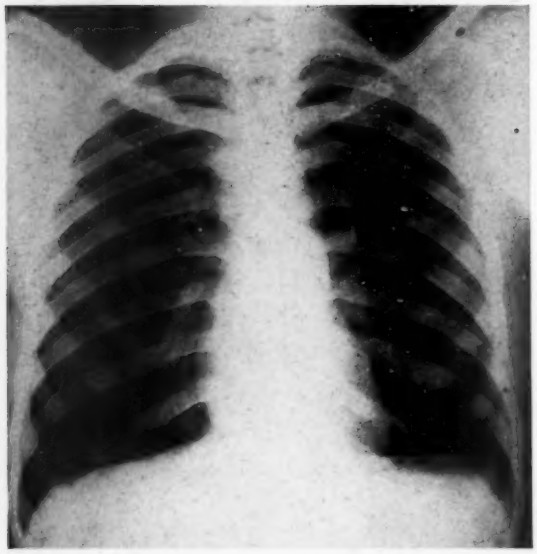

Fig. 3. Case 1. Shows the increase in the erosion of the fifth rib, which is now quite marked.

again demonstrated in the roentgen study made on September 11, 1947.

Comment. This case illustrates the resolution of the pulmonary involvement by coccidioides, but there was evidently a hematogenous spread localized to the fifth rib on the left side, as well as to various parts of the soft tissues, as this rib had undergone destruction. There was no bone production—only bone destruction. There was spread of the disease process from the bone to the overlying soft tissues with subsequent abscess formation, suppuration and ulceration.

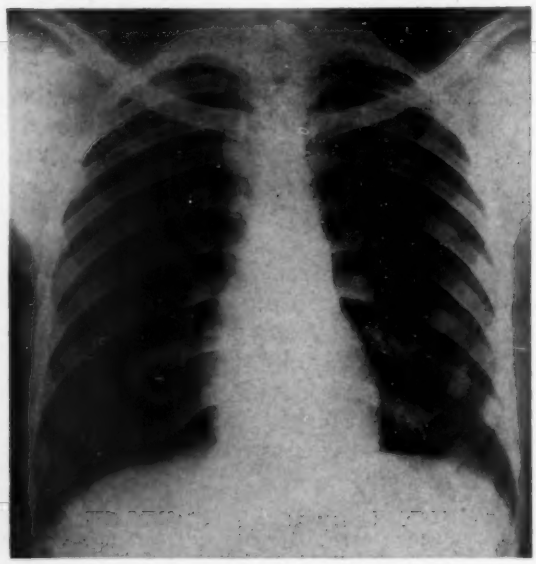

Fig. 4. Case 1. This roentgenogram illustrates the destruction of two-thirds of the anterior portion of the fifth rib on the left side.

Fig. 5. Case 1. Shows an area of necrosis and ulceration in the left lower chest, region of the fifth rib.

Case 2. H. W., a Negro male, aged twenty-one, who had been a resident of Kern County since 1926. History revealed that since the age of five years he had been having granulomatous lesions appearing in various parts of the body which would go on to abscess formation and suppuration. Serological tests had been positive. Patient was seen on September 9, 1947, complaining of pain in the lumbar area, of only four weeks' duration. Figure 6 reveals involvement of the anterior surfaces of the third, fourth and fifth lumbar vertebrae, which may well represent calcifications in the soft tissues.

Patient had multiple draining sinuses along the anterior and medial surfaces of the thighs, and there appeared to be some soft tissue calcification along the anterior surfaces of the third, fourth and fifth lumbar vertebrae (Fig.

6). It was noted that although this patient had areas of ulceration in the lumbar area posteriorly there was no involvement of the posterior surfaces of the lumbar vertebrae. Roentgen examination of the left femur (Fig. 7) showed some calcific densities in the soft tissues along the medial surface, but the femur itself was not involved. Figures 8 and 9 show areas of involvement in the lumbar area, and left thigh.

Fig. 6. Case 2. Shows the destruction of the anterior surfaces of the third, fourth and fifth lumbar vertebrae, with some sclerosis of the underlying bony structure in the fifth lumbar vertebra. There can be noted also some linear densities anteriorly which may represent calcification in the soft tissue.

Fig. 7. Case 2. Illustrates densities and linear streaking along the medial surface of the femur, suggesting calcification of the soft tissue. There was no invasion of the underlying bony structure.

Comment. This patient had been suffering from disseminated coccidioides for a period of sixteen years. During this time he had shown considerable resistance to the disease process as evidenced by the fact that the serological tests showed evidence of a moderately severe coccidioidal infection only. Evidently he was handling

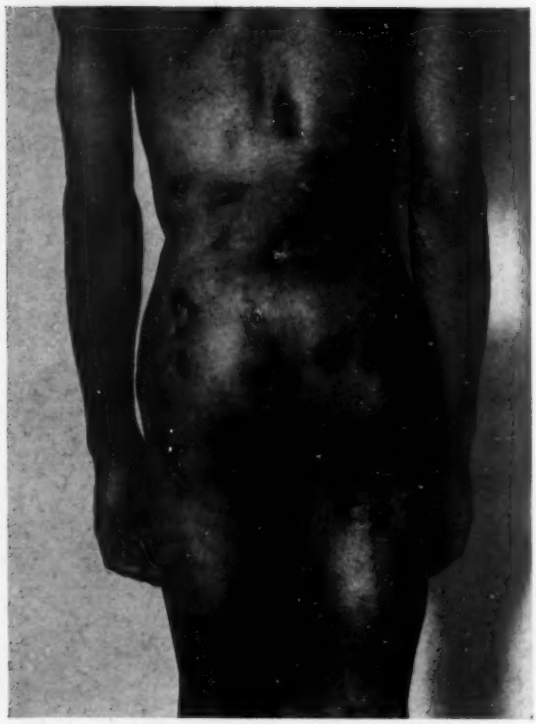

Fig. 8. Case 2. Shows numerous granulomatous lesions involving the lumbar area, the gluteal regions and the upper portion of both legs. Note the marked ulceration in the region of the fourth and fifth lumbar vertebrae.

Fig. 9. Case 2. Shows the granulomatous lesions along the medial surface of the left upper leg.

the infectious process fairly satisfactorily. This case illustrates the fact that while there were ulcerating granulomatous lesions involving the soft tissues, the underlying bone structure was not affected.

Case 4. I. A., a Filipino, aged thirty-three, who had been under treatment for pulmonary tuberculosis since 1939. He was first seen in Kern General Hospital on July 27, 1946, with chief complaints of frequency of urination, fever and nocturia of one month's duration.

Cystoscopic examination revealed findings suggestive of possible tuberculosis involving the bladder. Patient returned to the sanatorium.

Fig. 10. Case 4. Shows an osteolytic area in the medial portion of the navicular with breaking down of the margin. There is also noted marked calcification of the digital vessels.

He was next seen on January 13, 1947, in the Orthopedic Clinic with complaints of swelling of the medial and dorsal surfaces of the left foot. The swelling had been present for about six weeks before admission and had been increasing slowly until the time of examination. The swollen area extended from the toes to above the ankle and was somewhat tender on palpation and warm to touch. Roentgen examination of the foot (Fig. 10) showed a radiolucent area in the navicular bone, marked thinning of the cor-

tical bone, and a break in the cortex medially. There was also noted marked sclerosis of the digital vessels. Further roentgen studies, made on January 29, 1947, showed an increase in the destructive process involving the navicular bone.

On March 6, 1947, the navicular bone was excised.

Pathological report showed "considerable necrosis with extensive granulation tissue characterized by the formation of typical tubercles with Langhans' giant cells, epithelioid cells and surrounding rings of lymphocytes. There were occasional double contoured spherules seen which established the etiology of coccidioidal granuloma of bone."

On March 31, 1947, the patient complained of pain about the left knee. An area of demineralization and destruction of the cortical bone involving the patella was demonstrated on roentgen study (Fig. 11), with radiolucent areas in the lower end of the femur. These findings evidently represented a spread of the disease process to involve the patella and the lower end of the femur. There was a lapse of approximately three weeks from the time of operation and the findings in the left knee.

Comment. The patient was suffering from two lesions of a granulomatous nature—tuberculosis and coccidioidomycosis. The diagnosis of coccidiodomycosis was established by the findings of double contoured spherules in the sections taken from the tarsal navicular bone.

Fig. 11. Case 4. Shows bone destruction of anterior surface of patella, posterior surface of tibial plateau, and also bone destruction at the lower end of the femur.

Fig. 12. Case 9. Shows the marked destructive lesions involving the calvarium.

Fig. 13. Case 9. Shows the marked destructive lesions of the calvarium.

This illustrates the possible spread by surgical intervention, since experience with this disease shows that the best results are obtained by non-surgical methods.

Case 9. M. A. M., a white female, aged sixty-nine, who had been in Kern County for nine years. Admitted to the Hospital with a diagnosis of coccidioidal granuloma. History revealed that she had been ill for about five or six weeks previously and had been undergoing treatment in another hospital for lesions on the scalp. She had been discharged from the other hospital and had returned home when she developed a high fever, was mentally confused, and was then admitted to our Hospital.

Examination revealed an elderly female with slight mental confusion and multiple abscesses of the scalp. There was a granulomatous lesion on the right cheek and right side of the nose. The chest was negative on physical examination; however, roentgen studies of the chest showed a diffuse mottling throughout both lung fields which was diagnosed as coccidioidomycosis. Roentgen studies of the skull (Fig. 12 and 13) showed multiple areas of necrosis. Biopsies of these abscess formations on the skull revealed multiple coccidioidal endospores. The patient was skin tested and was negative, even in dilutions of 1 to 100.

Comment. This is the oldest case in our series. It is noted that in this instance the patient had had a negative skin test while suffering from a disseminated granulomatous lesion which, on biposy, demonstrated *Coccidioides immitis*.

SUMMARY

Seventeen cases of coccidioidal granuloma of bone are presented. These are proved cases seen at Kern General Hospital from July, 1942, to October, 1947. The colored race, Mexicans and Filipinos, are the predominating racial groups that have been shown to be most susceptible to the disseminated lesions. The lesions are seen most often in the third and fourth decades.

Roentgen findings are principally osteolytic, and occasionally there may be some sclerosis. However, in some of our cases there are suggestive linear calcifications in the soft tissues adjacent to bone. Our findings are at variance with those described by Carter, Benninghoven and Miller insofar as they describe bone involvements secondary to extensions from soft tissue granulomatous lesions. In none of our cases has a soft tissue abscess produced underlying bony invasion. On the other hand, the bone involvement may produce a soft tissue abscess with ulceration.

In many of our cases the primary lung involvement can no longer be demonstrated when the patients are seen with findings of a disseminated coccidioidomycosis.

A negative coccidioidin skin test may be of no, or little value, in making a diagnosis of osseous coccidioidal granuloma.

Serological examination is of diagnostic importance in determining the presence of a disseminated coccidioidomycosis.

Cultures, biopsies and tissues taken at autopsy are of importance in definitely establishing the presence of coccidioidomycosis.

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CALCIFICATION IN INTRASPINAL MENINGIOMAS

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ALTHOUGH abnormal calcification of the skull has long been of value to the roentgenologist in the diagnosis of intracranial neoplasms, it has rarely been helpful in the diagnosis of intraspinal tumors. Chief interest in the diagnosis of intraspinal tumors has been centered on erosion of the pedicles, increase in interpedicular distance, and changes in the intervertebral foramina, little attention being given to calcification within the vertebral canal.

Sporadic case reports have appeared in the literature concerning calcification within the spinal column seen on roentgenograms.[1,2,3,4,6,7,11,12,13,15] Dyke[8] referred to this calcification by saying: "When a definite, irregular mass of lime is seen within the spinal cord, the location and type of tumor has been determined, for in this clinic such an irregular mass of calcification has always proven to be within a meningioma." Brown[3] estimates that 15 per cent of intraspinal meningiomas are of the psammomatous or osteoblastic type, and that 4 per cent of these types should be visible on the roentgenogram.

It is the purpose of this paper to present 5 cases of intraspinal meningiomas which contained sufficient calcification to be visible on roentgenograms of the spine. No calcification was seen within any intraspinal tumor other than meningioma.

During the years 1937–1947, inclusive, there were 54 intraspinal neoplasms seen at the Buffalo General Hospital on the neurosurgical service of Dr. Wallace Hamby, 15 of which were meningiomas.

REPORT OF CASES

CASE I. R. C., a female, aged fifty-seven, noticed in 1929 that her feet felt numb and heavy, and that her steps had become awkward and jerky. This persisted until time of admission, in 1939.

On May 2, 1939, myelography of the spine showed an almost complete block to the column of lipiodol at the level of the sixth dorsal vertebra. Overlying the body of the fifth dorsal there was an oval, homogeneous density which appeared to be in the region of the vertebral canal. This could also be seen in the lateral projection. There were no bony changes in the vertebrae.

A dorsal laminectomy was done on May 3, 1939. A friable reddish-gray tumor, which contained an extensive calcium deposit on the right lateral surface of the tumor, was found. The upper pole of the tumor was partially covered by the lamina of the sixth dorsal vertebra.

Pathological Report. The tumor, which measured 3.2 by 1.5 by 1 cm, was grayish-pink in color and microscopically contained bone. Microscopic examination revealed a typical psammoma.

Note: Roentgenograms and pathological section were not available, so illustrations are not included.

CASE II. M. W., a white female, aged forty-three, since 1938 had noticed bilateral weakness in her legs. Since October, 1942, she has had low lumbar pain radiating down her legs, more on the left than on the right.

Myelography done on February 16, 1943, showed a complete block to the column of lipiodól between the tenth and eleventh dorsal vertebrae. Just above the block there was an irregular, flocculent type of density which could conform to calcification within a spinal cord tumor. This calcification, which was well seen on the lateral projection, was barely visible on the anteroposterior projection. There was also a slight widening of the interpedicular distance of the tenth dorsal vertebra, with some flattening of the inner aspect of the pedicle on the right side.

A dorsal laminectomy was done on February 23, 1943. A mass could be palpated through the dura, and this mass was so hard and fixed that it was impossible to introduce the rongeur blade between the dura and the lateral portions of the

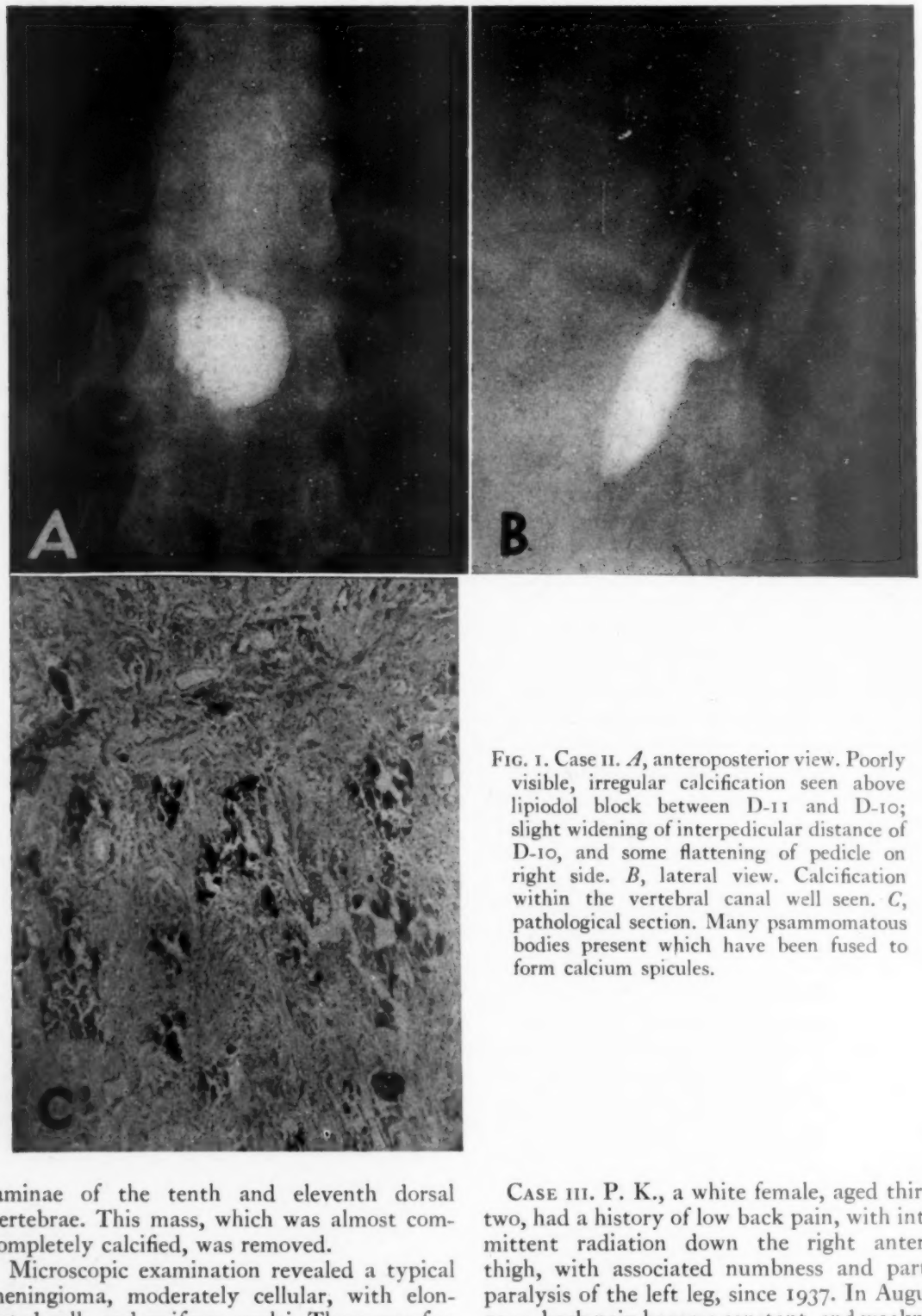

Fig. 1. Case II. *A*, anteroposterior view. Poorly visible, irregular calcification seen above lipiodol block between D-11 and D-10; slight widening of interpedicular distance of D-10, and some flattening of pedicle on right side. *B*, lateral view. Calcification within the vertebral canal well seen. *C*, pathological section. Many psammomatous bodies present which have been fused to form calcium spicules.

laminae of the tenth and eleventh dorsal vertebrae. This mass, which was almost completely calcified, was removed.

Microscopic examination revealed a typical meningioma, moderately cellular, with elongated cells and uniform nuclei. There was frequent whorl arrangement, with a great many psammoma bodies which had fused to form calcium spicules.

Case III. P. K., a white female, aged thirty-two, had a history of low back pain, with intermittent radiation down the right anterior thigh, with associated numbness and partial paralysis of the left leg, since 1937. In August, 1944, back pain became constant, and weakness of the left leg became pronounced. Since October, 1944, she has been unaware of the sensation of urination or defecation.

Roentgenograms on April 12, 1945, showed an oval-shaped area of homogeneous increased density in the vertebral canal at the level of the twelfth dorsal intervertebral space. This increased density was visible only in the lateral projection of the dorsolumbar spine. There was no increase in the interpedicular distance, no erosion of pedicles or enlargement of intervertebral foramina. Pantopaque spinogram showed complete block in the mid-portion of the first lumbar vertebra, with concave surface of pantopaque column at the block.

On April 24, 1945, a laminectomy was done. A pinkish-gray, friable, encapsulated neoplasm lying in the right anterolateral quadrant of the canal at the twelfth dorsal and first lumbar vertebrae was found, and removed.

Microscopic examination showed a moderately cellular meningioma, with frequent whorl arrangement. Numerous psammoma bodies were present, with distinct calcification.

Case iv. A. H., a white female, aged forty-six, began to complain of sensation of warmth and numbness in right foot and leg, in August, 1946, which gradually ascended to the hip and right half of abdomen and back. The left leg had a tendency to drag.

A pantopaque spinogram was done on November 11, 1946, which showed practically complete block at the level of the fifth dorsal vertebra; only a few drops of pantopaque passed this area. A tumor mass could be outlined on the right side of the canal, measuring approximately 2.5 cm. in length by 1.5 cm. in thickness. There was faint homogeneous calcification in this tumor mass. There was no change in interpedicular distance, no erosion of pedicles or enlargement of intervertebral foramina.

On December 12, 1946, a laminectomy was done. At the level of the fifth dorsal vertebra a small oval-shaped tumor compressing the cord was found, which was removed.

Microscopic examination revealed a highly cellular meningioma, with elongated cells with round and oval nuclei. The predominant feature in this tumor was the frequent whorl arrangement, with frequent psammoma bodies and moderate calcification.

Case v. E. R., a white female, aged fifty-two, with a history of intermittent pain in the right

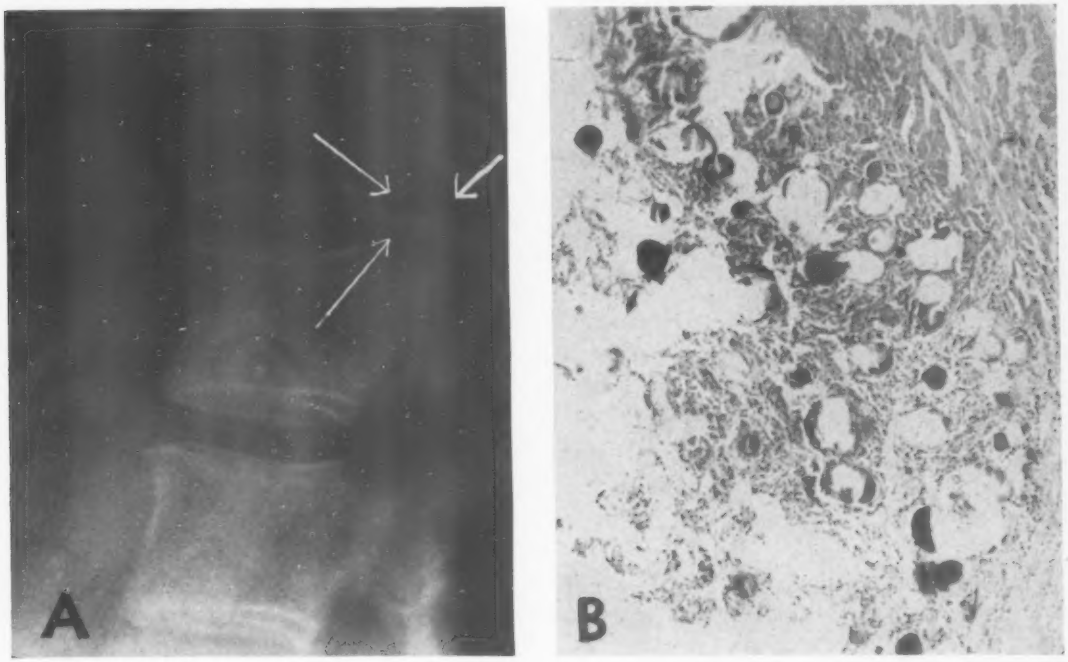

Fig. 2. Case iii. *A*, well defined oval mass of increased density at the level of the twelfth dorsal intervertebral space. This was not visible in the anteroposterior projection. *B*, pathological specimen. Numerous psammoma bodies with distinct calcification.

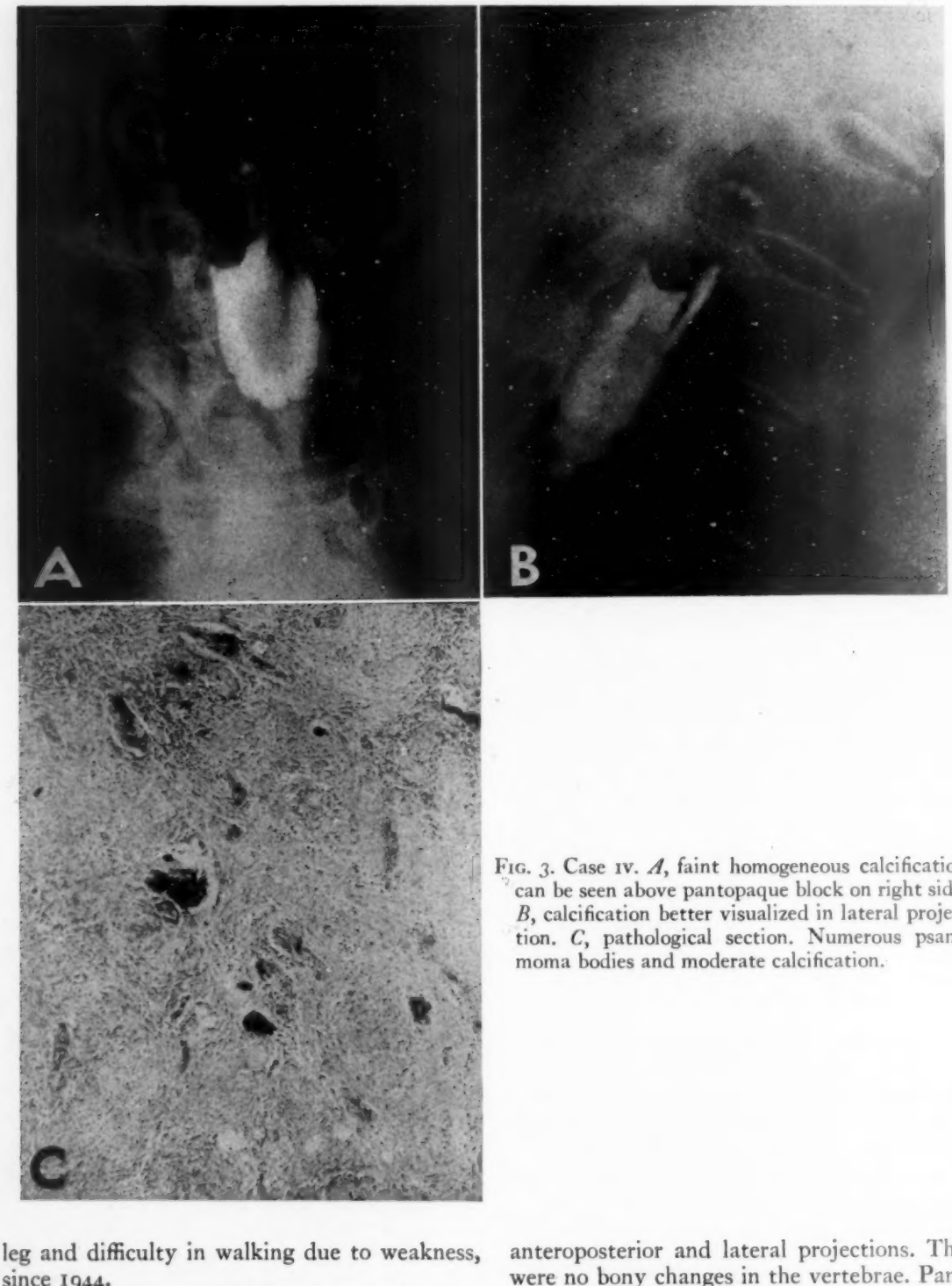

FIG. 3. Case IV. *A*, faint homogeneous calcification can be seen above pantopaque block on right side. *B*, calcification better visualized in lateral projection. *C*, pathological section. Numerous psammoma bodies and moderate calcification.

leg and difficulty in walking due to weakness, since 1944.

Roentgenograms of the dorsal spine, June 20, 1947, showed a calcified tumor lying in the region of the vertebral canal at the level of the sixth dorsal vertebral body. This measured about ½ inch in length, and was visible in both anteroposterior and lateral projections. There were no bony changes in the vertebrae. Pantopaque spinogram showed almost complete block to pantopaque at the lower border of the sixth dorsal vertebra. A small amount of contrast material passed the block. The block was at the lower level of the calcified tumor mass

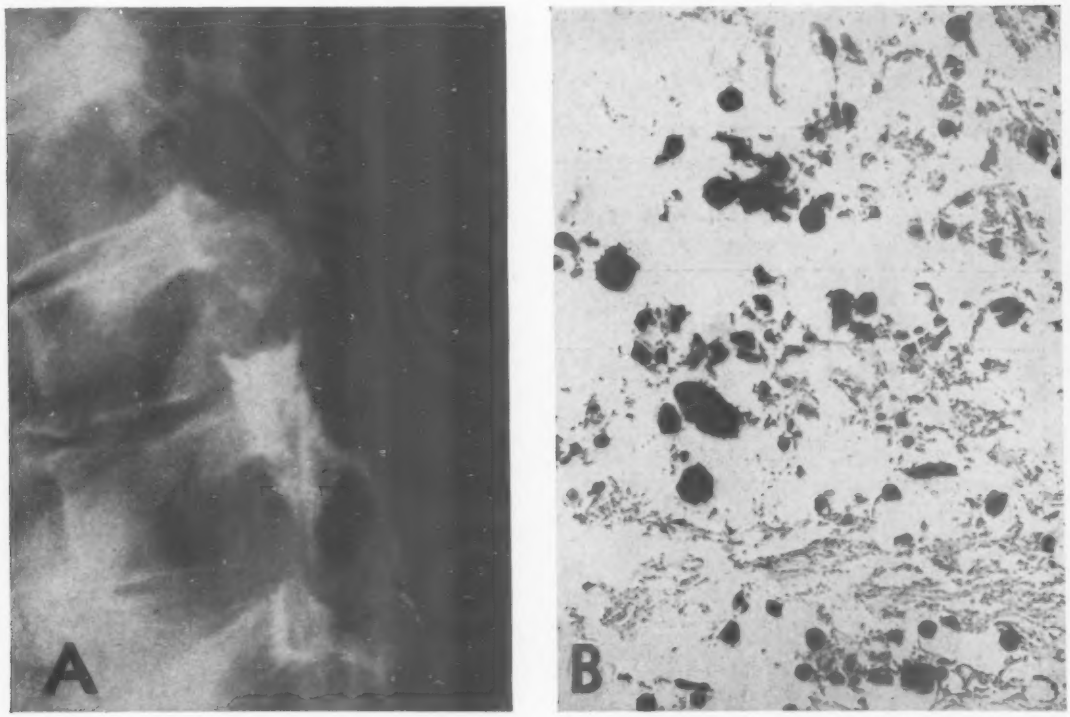

Fig. 4. Case v. *A*, calcified tumor seen above pantopaque block. *B*, pathological section. Many calcified masses scattered in the stroma.

which was seen on the plain roentgenogram.

Dorsal laminectomy was done, June 24, 1947. An extensively calcified tumor was removed from the left anterolateral aspect of the canal.

Microscopic examination revealed an ossifying meningioma with many bony trabeculae being laid down in a meningiomatous stroma. In some places there were calcified psammoma bodies scattered in the stroma. Another section showed only endothelial-like cells enclosing many markedly calcified bodies.

DISCUSSION

Extramedullary, intradural spinal tumors form 60 per cent of all spinal tumors, and intraspinal, extradural tumors constitute 20 per cent. Dyke[8] estimates that 25 per cent of extramedullary, intradural tumors may be localized roentgenographically by the changes they produce in contiguous vertebrae and surrounding soft tissues, while as many as 80 per cent of the extradural, intraspinal tumors will produce localizing changes. Camp[6] further corroborates this by stating that in his series of 164 cases of extramedullary spinal cord tumors, 29.8 per cent of these tumors caused secondary changes in contiguous vertebrae sufficient to permit roentgenological localization of the lesion.

Findings which are of aid to the roentgenologist in the localization and diagnosis of spinal cord tumors are: (1) erosion of pedicles and increased interpedicular distance, (2) bone destruction, (3) distortion of paraspinal soft tissues, (4) bone proliferation, (5) kyphosis and scoliosis, (6) calcification in a tumor. These changes have evoked much comment in the literature, notably by Elsberg and Dyke, and Camp.

Although the over-all incidence of diagnostic roentgen changes in spinal cord tumors is reported as high as 30.3 per cent, it is estimated that it is possible to make a roentgenographic diagnosis of spinal cord tumor in only 8 per cent of meningiomas.[8] In Camp's[6] series, 9.3 per cent of meningiomas produced changes in the vertebrae, and, in addition, 2 were calcified; thus,

13.9 per cent of meningiomas were diagnosed in this series.

TABLE I

ROENTGENOGRAPHIC CHANGES IN BONE

	Intradural Intraspinal Tumors	Extradural Intraspinal Tumors	Meningioma
Camp[6]	21.6%	47.2%	9.3*
Dyke	25.0%	80.0%	

* In addition, 2 were calcified—13.9 per cent.

Meningiomas constitute between 20 and 26 per cent of all spinal cord tumors (Table II), the greater majority of meningiomas being localized to the thoracic vertebrae (Table III).

TABLE II

Author	Spinal Cord Tumors	Meningiomas	Meningiomas per cent
Wolf	345	57	22
Elsberg	275	73	26
Rasmussen *et al.*	557	140	25
Camp[6]	198	43	22

A typical meningioma is a discrete encapsulated tumor, of an oval or spherical shape. The spinal, as well as the intracranial, meningiomas are believed to originate from clusters of arachnoidal cells. Microscopically, a meningioma consists of elongated fibroblastic cells with frequent whorl arrangement. Progressive degeneration with fibrosis, hyalinization and calcification of these whorls are common, resulting in the formation of psammoma bodies. In Wolf's series of 57 meningiomas, the psammoma bodies were present in 51; were abundant in 25; and in 6 tumors they were particularly abundant. Where these psammoma bodies are particularly numerous, they have a tendency to fuse and form calcium spicules. Actual ossification has been described in intraspinal meningiomas by Bailey and Bucy,[2] Brown,[3] Cushing and Eisenhardt,[7] Wolf[17] and Rogers.[16]

Calcification within spinal cord meningiomas is of three general types. The formation of numerous psammoma bodies results in a homogeneous increase in the density of the tumor, so that on the roentgenogram the tumor is visible only as an ovoid area of increased density. This type of calcification is rarely visible in the anteroposterior projection, but is discernible in the lateral projection of the spine. When the psammoma bodies are particularly abundant, numerous flecks of calcium are visible on the roentgenogram. If the psammoma bodies fuse to form calcium spicules, or actual osteoblastic activity takes place to form bone, a sharp, discrete, irregular mass of calcification may be seen on the anteroposterior and lateral roentgenograms.

A survey of the literature shows that calcification in a spinal cord tumor has proved to be within a meningioma, with rare exceptions. Gray[11] has reported a case showing calcification in the roentgenogram which was subsequently thought to be a vascular oligodendroglioma, or an hemangioblastoma. Rawling[15] has reported a case of teratoma which contained sufficient calcium to be seen on the roentgenogram.

There are probably several reasons why calcification in intraspinal meningiomas is not recognized more frequently on roent-

TABLE III

LOCATION OF MENINGIOMAS

Author	Total Meningiomas	Cervical	Thoracic	Lumbar	Lumbosacral
Elsberg	73	10	59	4	0
Brown	130	25	101	4	1
Rasmussen *et al.*	140	23	115	6	0

genograms, in spite of the fact that a high percentage are known to contain some calcium:

(1) The calcification frequently is not seen as a definite mass, but only as an area of slightly increased density because of the fine psammomatous type of calcification. The lateral views of the spine are frequently not scrutinized carefully enough for such minor changes.

(2) In other tumors the calcification is not sufficient to be visible. regardless of quality of roentgenogram or interpretation.

(3) The shadows of ribs superimposed upon the vertebral canal of the dorsal and upper lumbar vertebrae make visualization of small areas of increased density difficult.

(4) The technical difficulty in obtaining adequate lateral views of the upper three to four dorsal vertebrae, where so many meningiomas occur. Tomography may be of some value for better visualization of this region, in cases of suspected spinal cord tumors. Gray[11] reported a case in which the ordinary lateral and oblique views were not quite conclusive in proving the position of the calcification seen overlying the body of the third dorsal vertebrae in the anteroposterior view, but by tomography was able to prove that it lay within the vertebral canal.

SUMMARY

1. For all practical purposes, a calcified intraspinal tumor is a meningioma.

2. Out of a total of 15 meningiomas, five (or 33.3 per cent) contained sufficient calcium to show on the roentgenogram.

3. Homogeneous increase in density of a meningioma associated with psammomatous calcification will only be seen in a lateral roentgenogram of the spine. Probably more careful search for this type of calcification would result in an increase in the percentage of intraspinal meningiomas diagnosed preoperatively.

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SUPPLEMENT

The paper as presented above has undoubtedly given the impression that calcified spinal lesions are in most instances meningiomas. We had purposely excluded calcified herniated intervertebral discs, because previously such intraspinal calcifications had never offered any serious diagnostic problem. This is probably due to the fact that 90 per cent of disc protrusions occur in the lumbar area, and the majority of the remainder in the cervical region. Also, most reported cases of calcifications in intraspinal location in such cases have been in the low lumbar area. Our experience has been similar.

Calcification in the intervertebral disc has been described by Schmorl,[20] both in its normal position and in herniated position. He presented roentgenograms of anatomical specimens to illustrate this phenomenon. Brooke[18] presented a case in which a traumatic herniation of an ossified nucleus pulposus caused a complete transverse myelitis. At autopsy, flexion of the neck caused loose intervertebral substance to project 1 cm. into the spinal canal. He believed that, if roentgenograms of the neck had been taken in flexion, this ossified material might have been demonstrated. Cohen[19] reported a case of protruded intervertebral disc between the tenth and eleventh dorsal vertebrae. A review of the films after surgery showed what appeared to be a calcified mass between the tenth and

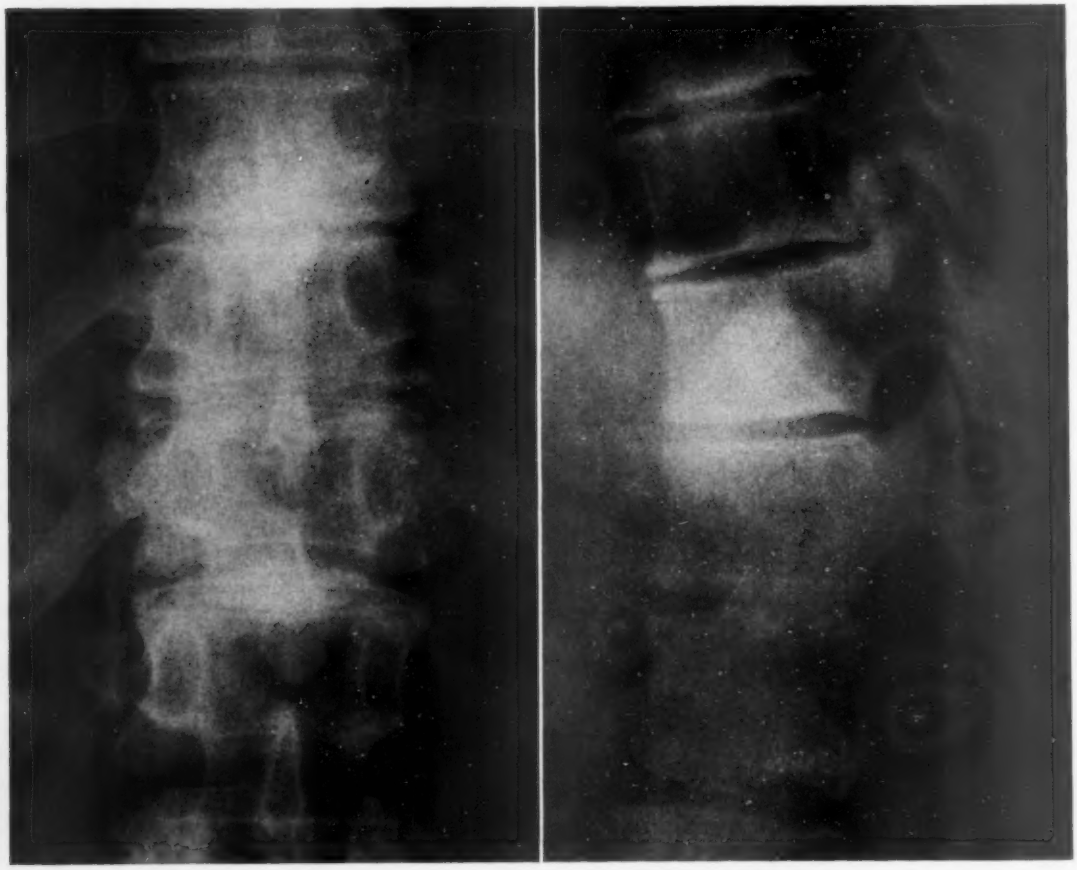

FIG. 5. Anteroposterior and lateral roentgenograms showing calcified intervertebral disc between D-12 and L-1 and the mass of calcification lying posterior to D-10 interspace.

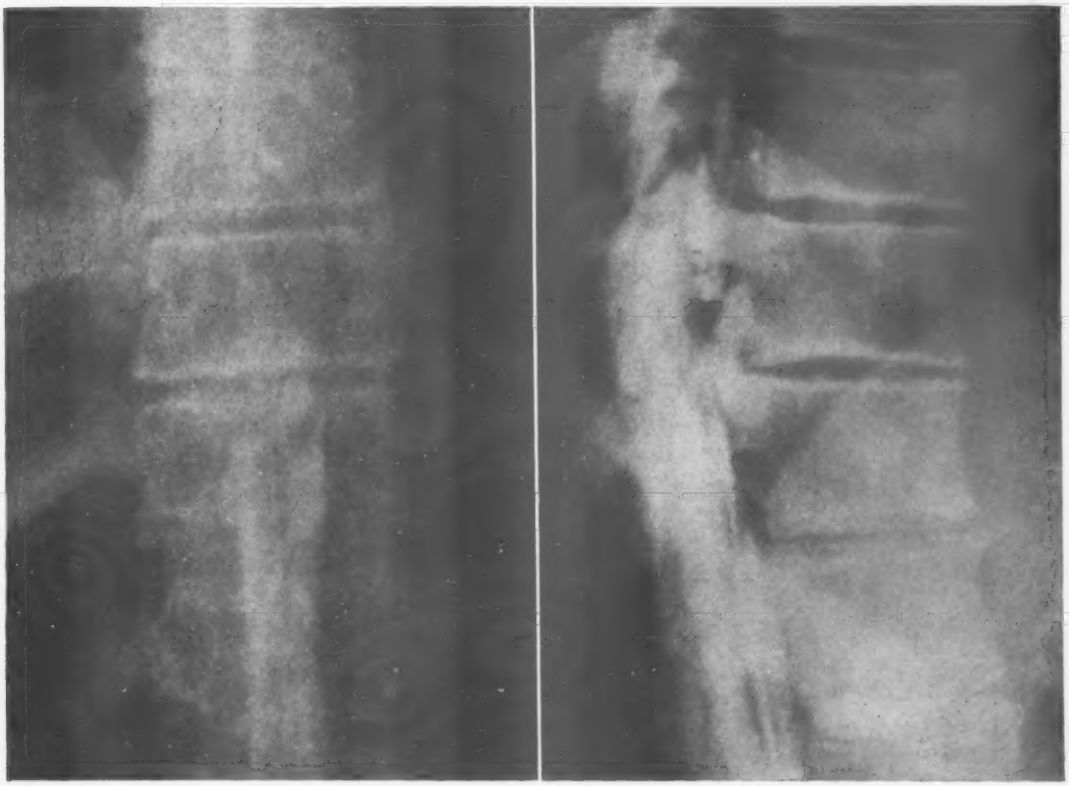

Fig. 6. Anteroposterior and lateral myelograms at same level as the plain films, showing type of displacement occurring. Note particularly the extent of the posterior displacement of the column above and below the lesion, as viewed laterally, rather than the intimate association of contrast material to the lesion which is usually encountered in intraspinal tumors.

eleventh dorsal vertebrae at the site of iodized oil arrest.

Since submission of the above paper, we have observed a case of intraspinal calcification in the dorsal area which occurred in a herniated intervertebral disc. We feel that the rarity of the lesion plus the necessity of including such lesions in a differential diagnosis warrants the inclusion of this case report.

CASE REPORT

Case vi. V. M., a male, aged fifty-five, who since 1940 had had numbness in the right foot ascending up the right leg, loss of pain and temperature sense in the right lower extremity, and pain in the lower dorsal area. The pain in the back was of a dull, aching nature, which radiated into the right upper abdomen and flank.

Deep reflexes were equal in the upper extremities. The knee jerk on the right was diminished, and the right ankle jerk was absent. Babinski responses were not present. There was hypalgesia and thermalgesia on the right, involving the right buttock and right leg. Vibration sense was diminished in the right ankle and leg. Motor function was good.

Lumbar puncture revealed initial pressure of 200 mm. Spinal fluid was clear. There was no evidence of subarachnoid block.

Plain roentgenograms showed a calcified disc between the twelfth dorsal and the first lumbar vertebrae. There was also a denser calcification lying posterior to the interspace between the tenth and eleventh dorsal vertebrae. This extended up and down the vertebral canal for about three-quarters of an inch, and was roughly triangular in shape. Pantopaque spinogram showed only a slight degree of block at the level of the calcified tumor. The pantopaque passed to the left and posterior to the calcified tumor. In the lateral projection the entire

pantopaque column appeared to be displaced posteriorly, and opaque material did not intimately surround the tumor mass. This type of deformity and lack of block was not what is commonly encountered in an extramedullary intradural tumor. This fact, combined with the type and extent of calcification immediately below, led us to consider the possibility of a protruded calcified intervertebral disc.

Decompressive laminectomy of L-1 and D-11, D-12 was done on March 22, 1948. An epidural, stony hard tumor was discovered anterior to the cord. The lesion could not be removed because of venous bleeding secondary to poor respiration. The operating surgeon was convinced that this was a calcified intervertebral disc.

Following surgery, a flaccid paraplegia, right Babinski response and sensory level at D-12 bilaterally was present. On discharge, there was some movement of the ankles in both lower extremities, bilateral Babinski response, flexion withdrawal at stimulation of the right leg, but no change in sensory findings.

DISCUSSION

This case has been added to the above paper for completeness. The occurrence of such lesions in the thoracic area, where most meningiomas occur, is certainly rare, but we now appreciate that they must be considered. Differential points are present which should in most instances establish the diagnosis. While a meningioma may be situated almost anywhere, nearly all herniations are noted opposite the intervertebral discs ventral or anterior to the subarachnoid space; rare exceptions are noted when a disc becomes detached and lies in the epidural space. When calcification is present in a herniated disc, the calcification is usually only a few millimeters in diameter, opposite the intervertebral space, ventral to the subarachnoid space; and in a fair percentage of cases there may be calcification in the portion of the disc remaining in the intervertebral space. With this calcification there may be some of the associated roentgenographic signs of intervertebral disc protrusion. There may be narrowing of the vertebral interspace, sclerosis of the adjacent vertebral spongiosa, eradication of normal curvature of the spine in the region of herniation, irregularity of the articular margins, proliferation of the articular facets, encroachment on the intervertebral wall by osteophytes. These signs are more common in the cervical area than in the thoracic or lumbar areas. A tumor may erode pedicles or vertebral bodies, and cause increase in interpedicular distance; a herniation never does. Studies with opaque contrast material will be of value in determining whether the calcified mass is extradural or intradural. Only a small percentage of meningiomas are extradural.

It would seem that the illustrations of Case VI (Fig. 5 and 6) will serve best to show the type of calcification and the myelographic changes to be expected in a calcified disc protrusion in the dorsal area.

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Vol. 62, No. 2

INTRACRANIAL CALCIFICATION IN ENCEPHALO TRIGEMINAL ANGIOMATOSIS*

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VASCULAR nevus of the face may be associated with a syndrome characterized by neurological signs referable to a lesion of the cerebral cortex and by an unusual type of intracranial calcification. This syndrome has been described adequately in the neurological literature but has received little attention in the roentgenological periodicals, despite the fact that the roentgenographic findings are often specific.

In 1879 Sturge[11] first described the occurrence of epileptiform seizures in association with vascular facial nevus and buphthalmos. Kalischer,[6] in 1897, described the necropsy findings in a similar case, demonstrating angiomatosis of the pia over one cerebral hemisphere. Several similar cases were reported in the following years, but it was not until 1922 that roentgenographic evidence of intracranial calcification in this condition was first noted by F. Parkes Weber.[12] Since that time well over one hundred cases of so-called Sturge-Weber (or Parkes Weber-Dimitri) syndrome have been reported showing various combinations of clinical and roentgenographic findings. The following case is reported to emphasize the clinical and roentgenographic findings considered typical of this condition.

CASE REPORT

S. S., female, white, aged twenty-nine, was admitted to the Psychiatric Service of Detroit Receiving Hospital because of auditory and visual hallucinations, and because she was violent and unmanageable at home due to irritability, lack of cooperation, and destructive tendencies.

The patient was born by breech delivery after a difficult labor. The mother noted a large "birthmark" of the upper right face and paralysis of the left arm and leg from birth. Development was retarded throughout childhood. The patient never attended school and was always shielded from other children. She could speak quite clearly and always recognized members of her family.

Family History. Essentially negative, especially as to cutaneous or neurological abnormalities.

Physical examination revealed a moderately obese female who exhibited definite evidence of mental deficiency. A large nevus venosum covered the right side of the face extending from the hair line to the mouth and from the ear to the midline anteriorly. The right side of the pharynx, uvula, and buccal mucous membranes were similarly involved. The nevus overlapped the midline slightly on the forehead and nose but terminated exactly in the midline of the lip and hard and soft palate. The tongue was uninvolved (Fig. 1). Ophthalmologic examination revealed only some pallor of the optic discs and increased ocular tension, interpreted as evidence of congenital glaucoma.

The left half of the body was definitely smaller than the right—the left arm being 3 inches shorter than the right and the left leg 2 inches shorter than the right (Fig. 2). Neurological examination revealed complete spastic paralysis of the left arm and spastic paresis of the left leg. Deep reflexes were uniformly hyperactive on the left with sustained ankle clonus on the left. The Babinski sign was positive bilaterally. There was no disturbance of the sensory modalities.

Laboratory Findings. Entirely negative, including negative serological findings. Permission for lumbar puncture refused. Electrocardiogram showed diffuse myocardial damage, evidenced by diphasic to inverted T waves in standard and precordial leads.

Roentgen Examination. Routine views of the skull showed a diffuse intracranial calcific density on the right outlining the convolutions of the cerebral cortex of the occipital and to a lesser degree the parietal lobes, the density being greatest posteriorly and fading out an-

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Fig. 1. Photograph showing right-sided distribution of facial nevus, limited chiefly to the area supplied by the ophthalmic and maxillary division of the trigeminal nerve.

teriorly as the central sulcus of Rolando was approached. The calcified cortex was separated from the inner table of the vault by a space of 1.5 cm., indicating cerebral atrophy (Fig. 3). Examination of the extremities showed obvious difference in size of the hands and of the feet, the left hand and foot being the smaller. The bones were well formed and exhibited normal architecture and density. Permission for cerebral pneumography and angiography was refused.

DISCUSSION

The foregoing case exhibited most of the classical features of the Sturge-Weber syndrome. In almost every case the facial nevus—a vascular nevus or "port-wine stain"—conformed to the area supplied by one or more main divisions of the trigeminal nerve, most often the ophthalmic. Symptoms usually began in infancy or early childhood with convulsions, either generalized or localized to the side contralateral to the facial nevus. These were described in about 80 per cent of the recorded cases. Mental retardation of varying degree was noted in about 60 per cent of cases. Paresis, almost always in the form of a spastic hemiplegia or hemiparesis on the side contralateral to the facial hemangioma, was also present in more than half

Fig. 2. Photograph demonstrating the hypotrophy of the extremities on the left.

the cases. Unilateral underdevelopment of the paretic side of the body may be noted as body growth progresses.

Various ocular abnormalities, among them buphthalmos, unilateral exophthalmos, ocular palsies, irregular pupils, optic atrophy, hemianopsia, and nystagmus, were described in over 30 per cent of cases. Rarer associated findings include speech defects and obesity.[4]

findings of the Sturge-Weber syndrome.[9]

The type of calcification noted in our case and in many of the most typical cases of this syndrome reported, however, consisted of a more diffuse calcific density outlining the contours of a portion of or the entire cortex of one cerebral hemisphere. The convolutional pattern was clearly defined in these cases. In all instances the involved portion of cerebrum appeared

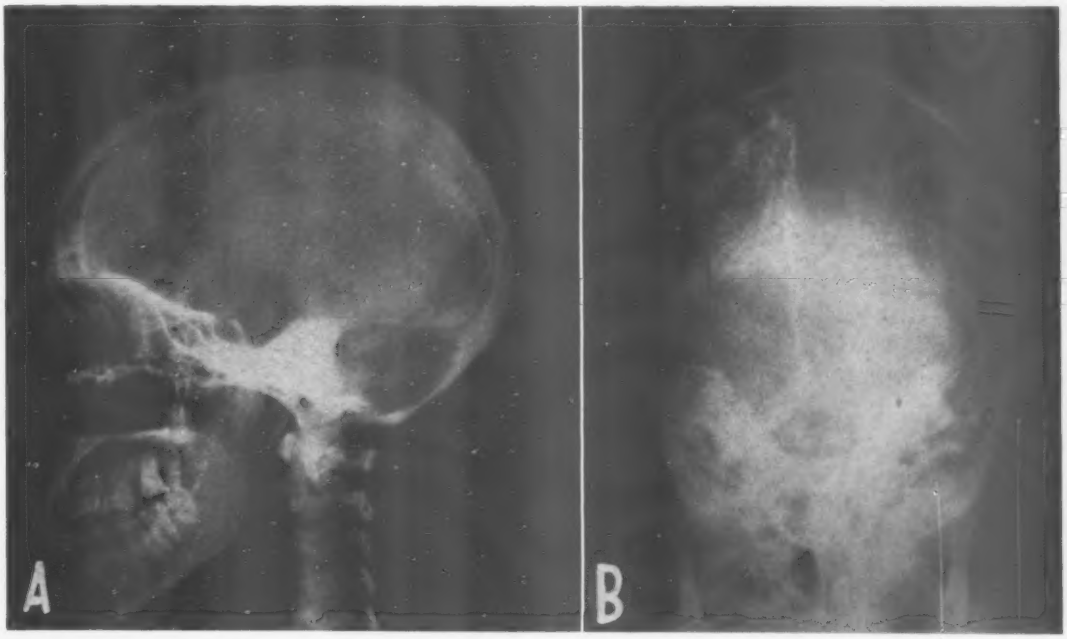

Fig. 3. (*A*) Right lateral roentgenogram of the skull of the same patient, showing calcification outlining the cerebral cortex. Separation of the calcified cortex from the inner table of the vault indicating cerebral atrophy is clearly shown. (*B*) Anteroposterior roentgenogram of same case showing limitation of the involvement to the right cerebral hemisphere. The characteristic convolutional pattern is again clearly seen.

The roentgenographic findings in Sturge-Weber syndrome are of primary importance since almost 90 per cent of the reported cases in which roentgenographic studies of the skull were obtained showed evidence of intracranial calcification.[4] Many of the cases exhibited the type of calcification known to be associated frequently with angiomata of the meninges—namely, sinuous, parallel linear streaks of calcification and, frequently, ring shadows apparently representing vessels seen end-on. This type of calcification is, of course, often seen in angiomata without the usual atrophic. In almost every case the involvement was posterior to the sylvian fissure and the central sulcus.

The basis for this unusual type of intracranial calcification is explained by the few cases of typical Sturge-Weber syndrome seen at necropsy or at craniotomy. In 1934 Krabbe[7] first correlated the roentgenographic findings with the neuropathology. He reported one case with diffuse intracranial calcification of the type described above which, at necropsy, showed only slight angiomatous formation of the pia anterior to the calcification seen on skull

roentgenograms, but corresponding exactly to the site of roentgenographic findings, there was marked deposition of fine granules of calcific material in the second and third layers of the cerebral cortex. This area showed definite cortical atrophy. Almost identical pathologic findings were reported by Stewart[10] in 1931 in a typical case of this syndrome, but no evidence of intracranial angiomata was found. Unfortunately, no roentgen studies were obtained in this case.

Actual angiomata of the meninges have been found at operation and at necropsy in some cases of this disease, but these cases usually have not shown typical diffuse calcification outlining the cerebral cortex. Green[3] reported a case in which sinuous, double-contoured calcification following the cerebral convolutions in the anterior parietal area were noted on the skull roentgenogram. A specimen obtained from this area by craniotomy revealed the presence of actual angiomata of the dura and pia arachnoid. There were some calcific aggregates in the leptomeninges as well as similar deposits in the outer laminae of the underlying cortex. In a few other cases[2,4,6,13] pathological reports cited the presence of angiomata or at least increased vascularity of the meninges in this condition, but no mention was made of the presence of calcification, either in the brain or its coverings.

Pneumoencephalography has been described in very few of the reported cases. Brock and Dyke[1] reported a case in which the air encephalogram confirmed the presence of cortical atrophy demonstrated by the diffuse cortical calcification. Definite internal hydrocephalus was also noted. In Green's case[3] the pneumoencephalogram revealed marked cortical atrophy of the involved hemisphere and dilatation of the corresponding lateral ventricle.

Cerebral angiography has also been reported in very few cases. In 1935 Egas Moniz and Almeida Lima[8] reported a typical case of Sturge-Weber disease in which they could demonstrate no association between the intracranial calcification outlining the surface of the occipital lobe and any of the vessels visualized on either the arterial or venous phase of cerebral angiography. Green,[3] however, in angiographic studies on his case, described involvement of the meningeal vessels in the region of the intracranial calcification.

Insufficient material on roentgenographic findings of pneumoencephalography and angiography in this condition is available to warrant a definite statement as to the findings to be expected. From a diagnostic standpoint, however, the routine skull roentgenograms showing intracranial calcification diffusely outlining the cerebral cortex, especially posteriorly, is considered to be pathognomonic, since we have been unable to find this type of calcification described as occurring in any other condition. Cerebral atrophy in the affected area can usually be made out without the use of contrast studies if the type of calcification just described is present.

An interesting phase of this subject is the apparently close association of encephalotrigeminal angiomatosis with certain other conditions which together have been grouped under the term "neurocutaneous syndromes." This group includes tuberous sclerosis, neurofibromatosis, and neuro-ocular angiomatosis (von Hippel-Lindau's disease) in addition to Sturge-Weber's disease. A common embryological background for these conditions has been postulated by Yakovlev and Guthrie.[14] These four syndromes bear some resemblance to one another in clinical picture and may exhibit some features in common. Intracranial calcification may occur in any of the neurocutaneous syndromes, but calcification conforming to the cerebral convolutional pattern is typical of encephalotrigeminal angiomatosis. The roentgenologic findings in these conditions have been described by Heublein, Pendergrass and Widmann,[5] and further discussion is beyond the scope of this presentation.

SUMMARY

1. A case of unusual intracranial calcification seen in association with ipsilateral facial nevus, contralateral spastic hemiplegia, and mental deficiency is presented.

2. The clinical, roentgenographic, and pathological findings in encephalotrigeminal angiomatosis are described. Two main types of intracranial calcification are seen in this condition—one type conforming to that seen in the meningeal angiomata, the other, probably characteristic of this syndrome, clearly outlining the convolutions of the cerebral cortex. The latter type is apparently produced by calcific deposits in the outer layers of the cerebral cortex.

3. The association of this condition with others of the neurocutaneous syndromes is discussed briefly.

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BIOLOGICAL EFFECTS OF ROENTGEN RAYS OF VARIOUS WAVELENGTHS

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IN order to compare the biological effects of different kinds of roentgen rays two series of experiments were performed. In the first series unfiltered, low voltage roentgen rays (soft roentgen rays), in the second series filtered, high voltage roentgen rays (hard roentgen rays) were used (Table I).

TABLE I

Kind of Roentgen Rays	kv. max.	ma.	Filter	Distance between Target and Object
Soft	43	3	—	17 cm.
Hard	140	3	3 mm. Al	17 cm.

Five hundred and ten adult tritons (*Triton cristatus;* Amphibia-*Urodela*) served as experimental animals. Their tails were treated locally with equal doses of both kinds of roentgen rays. Doses from 500 r to 10,000 r were administered in both series. The localization of treatment was accomplished with the apparatus shown in Figure 1 which consists of a wooden board (*A*) with sectors and a central disk (*B*) for restraining the animals, and a leaden collapsible cover (*C*) with openings in the central wall for triton tails. The bodies of 10 animals, bound with cheese cloth strips to the board, were protected with radiopaque cover while their tails, with the exception of 1 cm. of the proximal portion, were irradiated from the dorsal side. Approximately two months after the treatment the tails of all animals were amputated. Stumps, 2 cm. long, were left; thus the plane of the amputation passed through the treated region. The process of regeneration as well as some other phenomena would serve to characterize the degree of the biological effects of soft and hard roentgen rays.

The results of both series of experiments are given in Table II. The difference between them is rather insignificant. Thus from these experiments it appears that both soft and hard roentgen rays produce the same biological effect as concerns the mortality of animals, occurrence of necrosis, failure or presence of tail regeneration and the mean length of the regenerates. In both series the heaviest injuries, such as death, necrosis, and failure of the tail to regenerate were caused by large doses; tail regeneration occurred after the treatment with small doses and the length of the regenerates regularly decreased with the increase in dosage.

TABLE II

Kind of Roentgen Rays	Number of Animals in Series	Percentage of Animals Which Died during Experiment	Percentage of Animals Which Had Necrosis	Regeneration		
				Percentage of Animals without Tail Regeneration	Percentage of Animals with Tail Regeneration	Mean Length of Tail Regenerates in mm.
Soft	250	49.5	36.4	62.0	38.0	4.95
Hard	260	41.5	39.0	63.5	36.5	4.23

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These results agree with those of many other authors, e.g., Wood,[36] Packard,[25,26,27] Piepenborn,[29] Vierheller and Saralegui,[33] Hoffmann,[14] Langendorff, Langendorff and Reuss,[19] Obreschkove,[24] Dahl,[6] Marshak,[22] del Buono,[4] Bade,[1] Fabergé,[10] Lasnitzki and Lea,[20] Lingley, Gall and Hilcken,[21] Pascher and Kanee,[28] and others.

experiments analyzed in this paper have to be described once more. If the results of the treatment with both kinds of roentgen rays are considered from the standpoint of all features used above no significant difference can be noticed. But if another sign is taken as characteristic in the evaluation of soft and hard roentgen-ray effects,

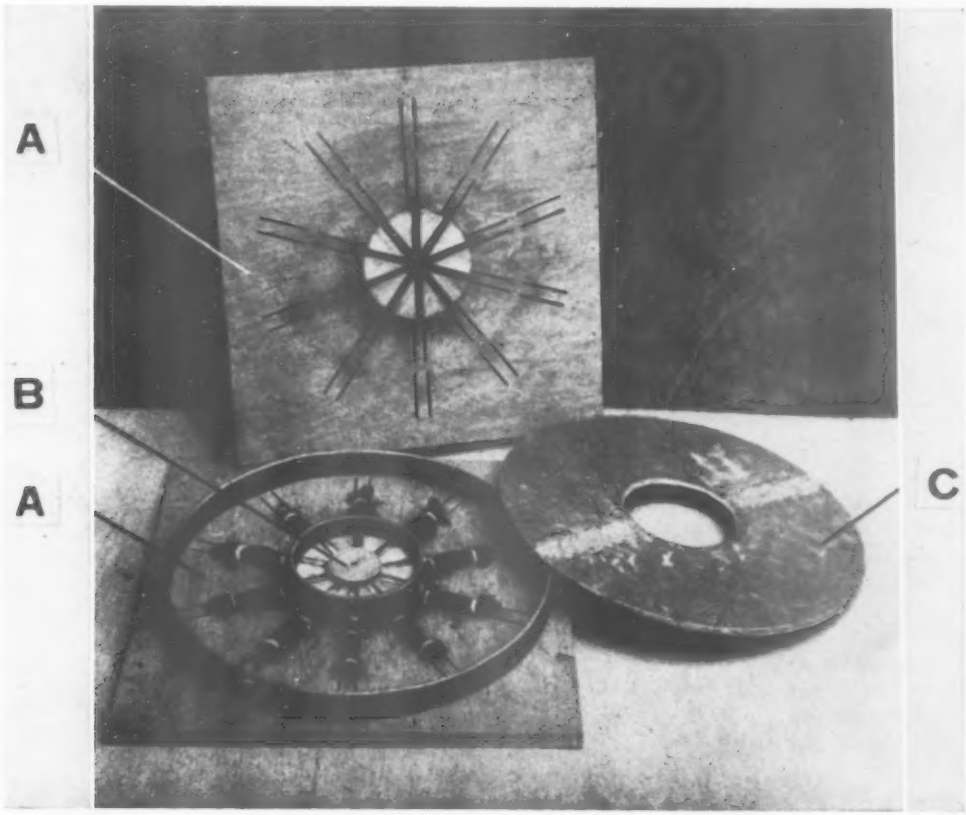

Fig. 1. The apparatus for local irradiation of triton tails.

But there is another opinion according to which these two kinds of roentgen rays produce different results, e.g., Rost,[31] Holthusen,[15] Martius,[23] Wintz and Rump,[35] Lachmann and Stubbe,[18] Ingber,[16] Dognon,[8,9] Henshaw and Francis,[13] Goldfeder,[11] Haberman and Ellsworth,[12] den Hoed and Stoel,[7] Juris,[17] Roffo,[30] Weiss,[34] and others.

When comparing the effects of soft and hard irradiation, the kind of treated material, its size, and the method of interpreting the effects have to be taken into consideration. In order to explain this statement the namely the morphological structure of regenerates, other conclusions ought to be drawn.

In tritons the normal tail regenerate is a symmetrical organ divided by the axial skeleton into equal dorsal and ventral parts. This symmetry is conserved even in cases where the plane of the amputation is not perpendicular to the long axis of the tail (Barfurth[2]). The regenerates of the animals treated with hard roentgen rays had symmetrical structures in 93 per cent of the total number of cases. As to the

regenerates of the animals treated with soft roentgen rays only 41 per cent of them were symmetrical while 59 per cent were asymmetrical. Twenty-eight per cent of regenerates had a strongly expressed upward curvature of the growth axis (Fig. 2).

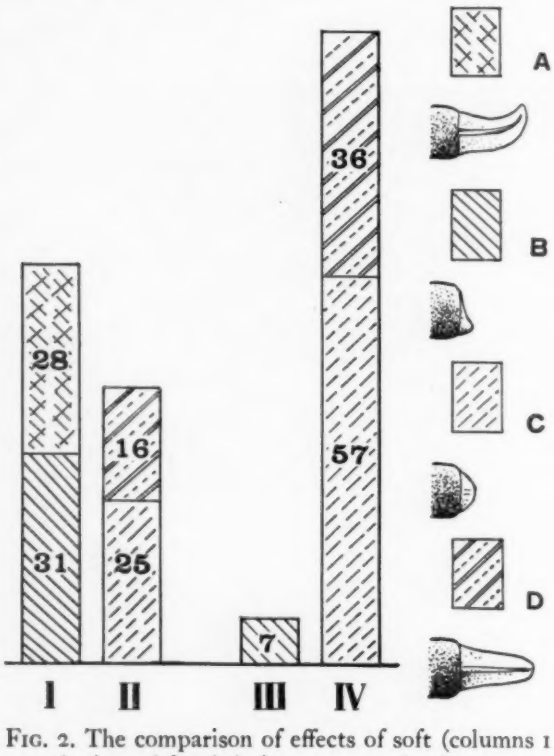

Fig. 2. The comparison of effects of soft (columns I and II), and hard (columns III and IV) roentgen rays on the morphological structure of tail regenerates.
A, large regenerates with clearly expressed curvature.
B, small regenerates with asymmetrical structure.
C, small regenerates with symmetrical structure.
D, large regenerates with symmetrical structure.

In *Triton cristatus* this phenomenon had been observed and described by Brunst and Chérémétieva.[3] Similar asymmetrical forms were observed for the first time in tail regenerates of tadpoles of *Pelobates fuscus* treated with soft roentgen rays (Scheremetjewa and Brunst[32]). At that time a question arose as to why the asymmetry was invariably the same; the axial part of the regenerates was always turned upward instead of being directed horizontally. The theoretical suggestions that this curvature was caused by the direction of the roentgen rays was demonstrated by experiments in which the irradiation with the same kind and dose of roentgen rays was applied not from the dorsal but from the ventral side. All curvatures were directed downwards (Chérémétieva and Brunst[5]).

The beam of unfiltered roentgen rays used in the "soft series" contained various kinds of soft rays. According to Wucherpfennig[37] such a beam of roentgen rays passing through the water gradually loses a considerable part of the "softest" rays. This is due to the absorption of these "softest" rays by the water. The originally applied dose decreases in proportion to increase in the depth of the water (Table III).

Table III

Depth in mm.	Percentage of the Dose
1	100
2	90
3	80
4	70
5	60
6	50
7–8	40
9–10	30

All living tissues possess almost the same absorption ability. They act like a filter. Therefore the part of the treated organ situated closest to the roentgen tube gets the dosage indicated, while other parts approaching the opposite side receive a greatly decreased dosage. This gradual decrease in dosage corresponding to the depth, resulted in the unequal inhibition of the regenerative capacity in different parts of the organ. This undoubtedly caused the formation of asymmetrical regenerates. Such is the case with soft roentgen rays. The hard roentgen rays used in the experiments were homogeneous, as they were filtered. They passed through the whole thickness of the tissues almost without suffering any loss through absorp-

tion and no obvious differences could be noticed between the acting doses at the opposite sides of the tails 10 to 12 mm. high. That was why only 7 per cent of regenerating tails were inhibited asymmetrically (Fig. 2).

SUMMARY

These experiments show that some biological effects of the soft and hard roentgen rays are apparently identical, e.g. the mortality of treated animals, frequency of necrosis, absence and presence of tail regeneration and the mean length of regenerates. But as regards the structure of the regenerates, there is a great difference between effects of soft and hard roentgen rays as a result of the absorption of soft rays by tissues. The last becomes especially evident if the irradiated object has a considerable thickness.

We wish to express appreciation to Mr. Donn Eric Rosen for help in the preparation of the manuscript.

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DEPTH DOSE DATA AND ISODOSE DISTRIBUTIONS FOR RADIATION FROM A 22 MEV. BETATRON

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INTRODUCTION

IN THE last few years several papers[1,2,4] have been published on the possible uses of the high energy roentgen radiation from the betatron for medical therapy. Before such radiation can be used effectively for this work, problems involving stray radiation, the collimation of the beam, the production of fields of a variety of different sizes and the construction of a betatron which is flexible with respect to its position and orientation of beam must be solved. Recently the betatron group of the University of Illinois[1] has published information on the first two problems stated above. The authors are indebted to them for many of the ideas discussed in this paper. The authors have produced fields of radiation of different sizes and have found how the depth dose depends on area and focal skin distance. The problem of flexibility of the betatron beam still remains to be solved. By using the information which has been obtained by Quastler and Clark[5] on the biological effectiveness of betatron radiation it should be possible to employ the betatron for roentgen therapy.

EXPERIMENTAL ARRANGEMENT FOR DEPTH DOSE MEASUREMENTS

Part of the apparatus used for the measurement of the distribution of radiation within a scattering medium is shown in Figure 1. The phantom consisted of a tank of water as illustrated. Radiation from the betatron was introduced into this tank of water through an aluminum window 0.4 mm. thick. The ionization chamber was connected to a pre-amplifier as indicated and arranged so that it could be moved in a controlled and known way in three directions mutually at right angles. This was done very conveniently from the control room by means of two pairs of selsyn generators and motors. The selsyn motors were used to drive screws of 1 mm. pitch. In the diagram of Figure 1 these are shown directly connected to the screws, but in actual fact they were connected to the screws by means of a small chain and sprocket. The selsyn generators in the control room were turned by hand and the position indicated by means of a small counter above each selsyn. Since one turn of the selsyn generator corresponds to one turn of the selsyn motor, which in turn corresponds to a motion of 1 mm., the position indicators gave the position in millimeters. The third motion was obtained by means of a pinion and ratchet arrangement which is not shown in Figure 1. This arrangement has proved useful for making routine measurements of radiation in hospitals. Where a vertical roentgen-ray beam can be used the radiation is made to impinge on the surface of the water in the tank.

The ionization chamber and pre-amplifier were connected by a long flexible cable to a sensitive negative feedback D.C. amplifier. This amplifier was developed by some of the authors.[3] The details of construction may be obtained from the original paper. To make measurements in this phantom it is necessary continually to monitor the roentgen-ray beam from the betatron. This can be done in several ways but one simple way which proved to be satisfactory was to place a large monitor ionization chamber as indicated in Figure 1.

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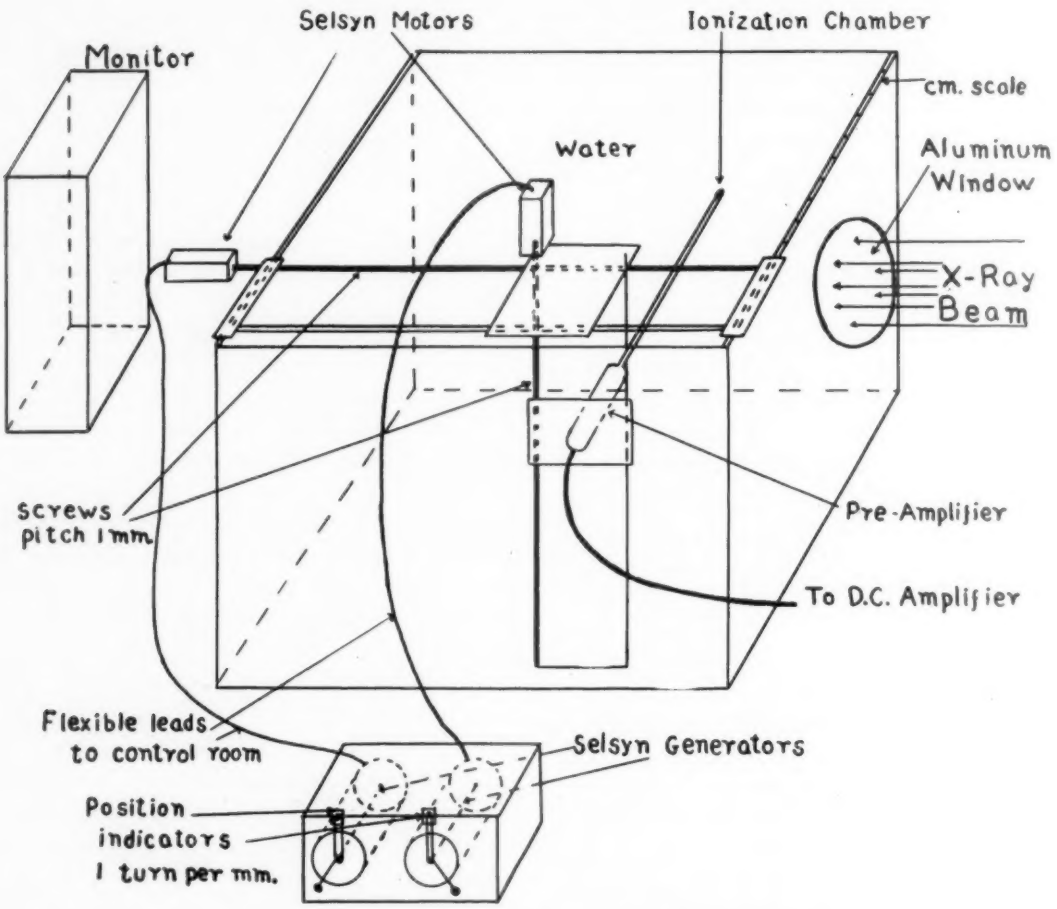

FIG. 1. Schematic diagram of water phantom showing the method for remote control of the position of the probe.

This chamber was provided with fifteen thin lead plates and was connected to the standard amplifier provided with the Allis-Chalmers betatron. This ionization chamber actually measured the amount of radiation which passed through the tank of water. For all practical purposes, this will be independent of the position of the probe.

The details of the thimble ionization chamber and its connection to the pre-amplifier are shown in Figure 2. The details of the pre-amplifier will be found in the

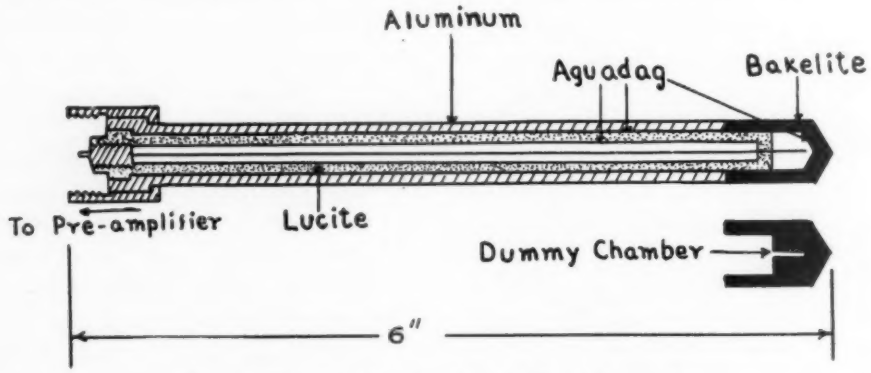

FIG. 2. Diagram showing the details of the probe.

original paper.[3] Considerable difficulty was experienced in producing a connection between the pre-amplifier and the thimble chamber which was not sensitive to betatron radiation. To investigate this effect, a probe assembly was made using a dummy ionization chamber (see Fig. 2) and this assembly was placed in the betatron beam. It was found that a stem of very thin walls gave a positive deflection on the amplifier. The polarity of the D.C. amplifier was such that a positive deflection corresponded to the collection of positive charges on the central electrode. As the thickness of the aluminum wall was increased, or a sleeve of aluminum was placed on the outside of the stem, the positive deflection was decreased and finally reversed to give a negative deflection. The authors are as yet unable to explain this phenomenon. However, by trial and error it was possible to construct a probe assembly with a dummy ionization chamber which gave practically no deflection under the action of the betatron radiation. To make sure that this neutralization was present during the experiments with the betatron, the following procedure was carried out. After a set of measurements had been made using a real ionization chamber on the end of the probe, the dummy cap was placed over the probe and the probe was placed in the position at which the original measurements had been made. In all cases it was found that the effect of the radiation on the stem was of the order of only 1/1000 to 1/5000 of the effect on the ionization chamber. The ionization chamber used had a volume of 200 cu. mm. If a smaller ionization chamber had been used the effect of the radiation on the stem would have been appreciable.

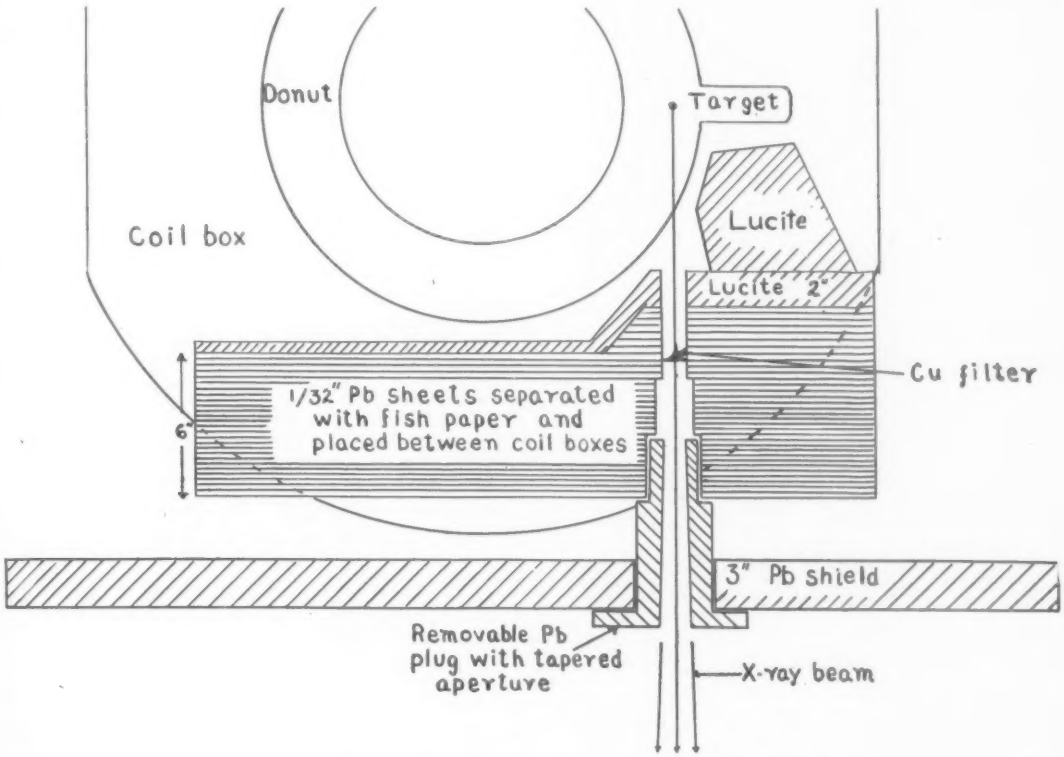

Fig. 3. Schematic top view of the betatron showing the collimating and shielding arrangements, etc.

In making measurements the betatron was adjusted until a pre-selected intensity of radiation was indicated by the monitor and the reading of the D.C. amplifier was taken when the betatron gave this output.

Little difficulty was experienced in holding the betatron output constant at the preselected value enough to get a reading on the D.C. amplifier.

SHIELDING AND COLLIMATION PROCEDURE

The arrangement of the betatron and its associated shield is shown diagrammatically in Figure 3. Many of the features included in the shielding process are taken from the work of the Illinois group.[1] The main shield consisted of laminated sheets of lead separated with fish paper placed between the coil boxes of the betatron with the dimensions shown. Next to the donut was placed a layer of lucite which served to stop stray electrons without any appreciable production of roentgen radiation. The hole through the lead shield consisted of a series of square openings as indicated. After this shield was mounted it was found that there was considerable radiation escaping just above and below the shield. To reduce this radiation and other scattered radiation a 3 inch lead shield was placed across the front of the betatron and a square hole was cut in it in line with the holes in the inner lead shield. It was found possible to insert through this lead shield and into the inner lead shield any one of a series of hollow lead plugs, one of which is shown in Figure 3. The holes in these plugs were circular in cross section and tapered to give fields of diameters 4, 6, 8 and 10 cm. respectively at a distance of 70 cm. from the focal spot. With such an arrangement it was found that radiation at all points in the betatron room was reduced to a satisfactory level. Such an arrangement offers many advantages for nuclear physics experiments since plugs with smaller tapered holes can be inserted at will to produce a very finely collimated roentgen-ray beam.

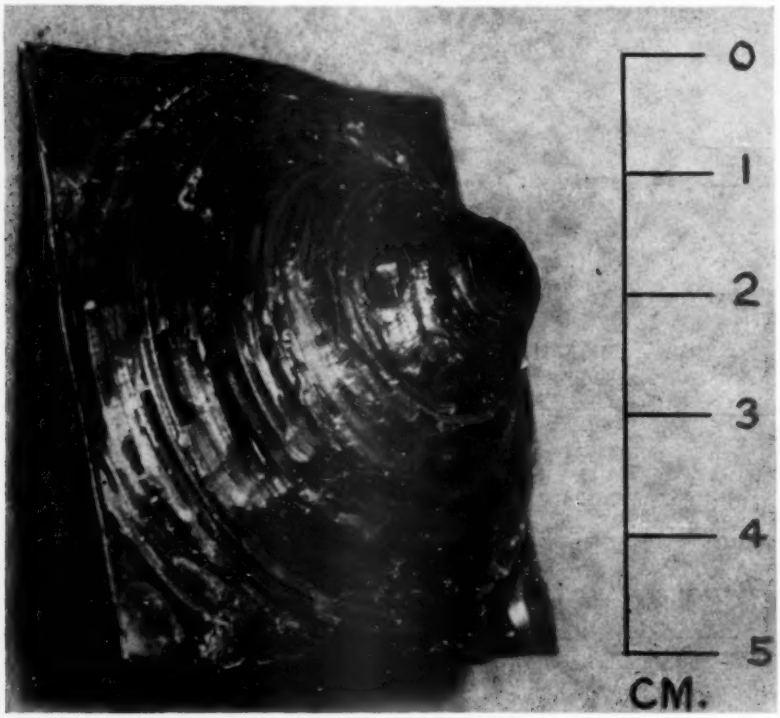

FIG. 4. Photograph of the copper compensating filter.

It is well known that the radiation from the betatron is projected primarily in the forward direction, and beyond small angles to this direction the radiation intensity falls off very rapidly. To achieve a useful

beam for therapeutic purposes a compensating filter was developed. The authors found that a copper filter was more easily constructed than one of carbon,[1] and gave results which were just as good. An analysis of the total absorption coefficient will show that copper has almost a constant absorption coefficient from 5 to 25 mev., so that the use of such a filter will not change the quality of the beam. The copper filter was placed far enough back in the shield to assure that the fringing field of the betatron would divert from the beam the electrons ejected from the filter. A photograph of the copper filter is shown in Figure 4. It was found necessary to make the filter somewhat asymmetrical due to the nature of the beam of radiation from our betatron. The shape of the copper filter was determined by trial and error, using the D.C. amplifier and ionization chamber for checking the variation of the intensity over a cross section of the beam and photographic films for the detection of sudden changes in intensity. By this process it was possible to obtain the distribution of radiation shown in Figure 12. The authors feel that this distribution is very satisfactory from the point of view of therapy. In fact, the falling off of the beam as one proceeds laterally from the axis of radiation is less pronounced than is the case with a low voltage roentgen-ray distribution. It will be seen that for a 10 cm. circular field, the 100 per cent isodose curve reaches to within 1 cm. of the edge of the beam.

RESULTS OF DEPTH DOSE MEASUREMENTS

Measurements of the depth dose were carried out for circular fields of diameter 4, 6, 8 and 10 cm. at 70 cm. focal skin distance and 6, 9, 12 and 15 cm. at 105 cm. focal skin distance. In all cases the doses were expressed as a percentage of the maximum dose. Typical results for two fields are shown in Figure 5 and a summary of all the results are given in Table 1. It will be seen that the maximum dose occurs 4 cm. from the surface and that the percentage depth dose rises rapidly from a small value at the surface to this maximum and then falls off much more slowly. It will be seen that the variation from the average curve does not exceed 1 per cent. Due to the thickness of the aluminum window in the phantom and the finite size of the ionization chamber, measurements could not be made with the center of the ionization chamber closer than 0.5 cm. to the surface. Theory indicates that the percentage depth dose at the surface should be zero, while extrapolation of the graphs in Figure 5 to zero depth indicates that the percentage depth dose at the surface is certainly less than 30 per cent. From Table 1 it will be seen that for small depths some variation in

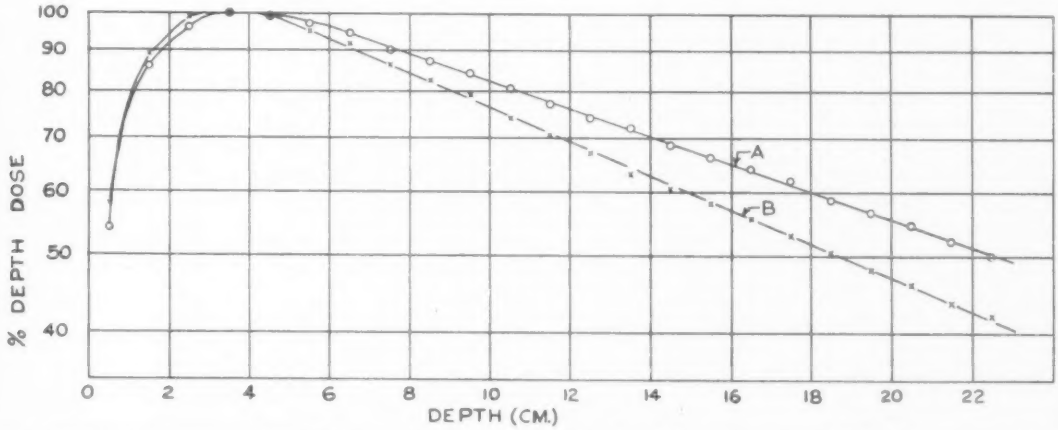

Fig. 5. Typical percentage depth dose graph: *A*—15 cm. circular field at 105 cm. F.S.D.; *B*—10 cm. circular field at 70 cm. F.S.D. using radiation from a 22 mev. betratron with a copper compensating filter.

dose occurs from one size field to another. This is due to the difficulty of making accurate measurements in this region where the depth dose changes rapidly with depth.

From Table 1 it is apparent that for a given focal skin distance the percentage depth dose along the axis is independent of the area of the field. This is in sharp contrast with the variations observed with standard therapy radiations. For this reason the average percentage depth doses (P_{70} and P_{105}) for each depth studied were calculated and are recorded in Table 1 for both focal skin distances. These average depth doses have been plotted and appear in Figure 6. That the percentage depth dose is independent of area is due to the fact that electrons are projected in the forward direction by the action of the high energy photons. The actual percentage depth doses herein reported are considerably greater than those obtained by Charlton and Breed[2] who obtained maximum ionization for 20 mev. radiation at a depth of 1.5 cm. but are in essential agreement with those obtained by the Illinois group.[1]

TABLE I

DEPTH DOSE DATA FOR 22 MEV. ROENTGEN RAYS AT 70 AND 105 CM. FOCAL SKIN DISTANCE USING A COMPENSATING COPPER FILTER IN THE ROENTGEN-RAY BEAM

Depth (x)	F.S.D.—70 cm.					F.S.D.—105 cm.					Universal Depth Dose
	Diameter of circular field				Average Value	Diameter of circular field				Average Value	$A=P_f\left[\frac{f+x}{f+4}\right]^2$
	4 cm.	6 cm.	8 cm.	10 cm.	P_{70}	6 cm.	9 cm.	12 cm.	15 cm.	P_{105}	
0.5	50	50	51	53	51	48	48	50	53	49.5	46
1	72	70	70	72	71	68	70	70	74	70	66
2	94	92	92	94	93	88	90	90	91	90	87.5
3	99.5	99	99	99.5	99	98	98	98	98	98	96.5
4	100	100	100	100	100	100	100	100	100	100	100
5	98.5	98	98.5	98	98.5	99.5	99.5	99.5	99	99.5	100
6	94	93.5	94	93	93.5	97	96	97	96.5	96.5	99
7	89.5	89	89.5	88.5	89	93	92.5	93	92.5	93	97
8	85	84.5	85	84.5	85	89	88.5	89.5	89	89	95
9	81	80.5	81	80.5	81	86	85.5	86	86	86	93
10	77	77	77	76.5	77	82.5	82	83	82.5	82.5	91
11	73.5	73	73.5	73	73.5	79	78.5	79.5	79	79	88.5
12	70	70	70	70	70	76	75.5	76.5	76	76	86.5
13	66.5	66.5	66.5	66.5	66.5	73	73	73.5	73	73	84.5
14	63.5	63.5	63.5	63.5	63.5	70	70	70.5	70	70	82.5
15	60.5	60.5	60.5	60.5	60.5	67	67	68	67.5	67.5	80.5
16	57.5	57.5	57	57	57.5	64.5	64.5	65	64.5	64.5	78.5
17	55	55	54.5	54.5	55	62	62	63	62.5	62.5	76.5
18	52	52	52	52	52	59.5	59.5	60.5	60	60	75
19	49.5	49.5	49.5	49.5	49.5	57	57	58	57.5	57.5	73
20	47.5	47.5	47	47	47.5	54.5	55	55.5	55	55	71.5
21	45	45	44.5	44.5	45	52	52.5	53.5	53	53	69.5
22	43	43	42.5	42.5	43	50	50.5	51.5	51	51	68

Because the depth dose is independent of area the authors have made an effort to analyze the curves shown in Figure 6 to see how these depend on focal skin distance and depth. An analytical expression which fits the data reasonably well can be obtained as follows.

Let I_0 represent the flux density of the incident photons at the surface of the phantom (see Fig. 7). At a depth z below the

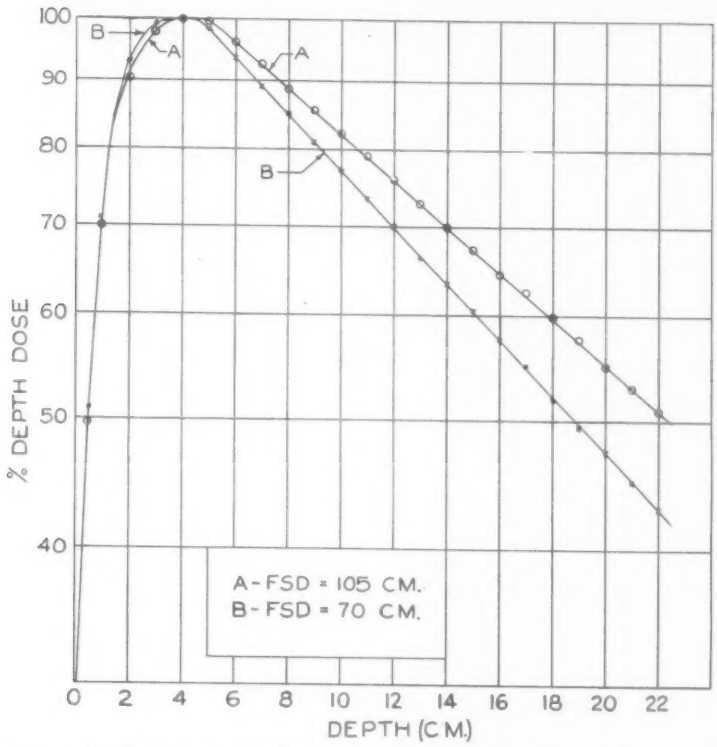

FIG. 6. Average percentage depth dose graphs for focal skin distances of 70 and 105 cm. using radiation from a 22 mev. betatron with a copper compensating filter. The points shown are the average values recorded in Table 1.

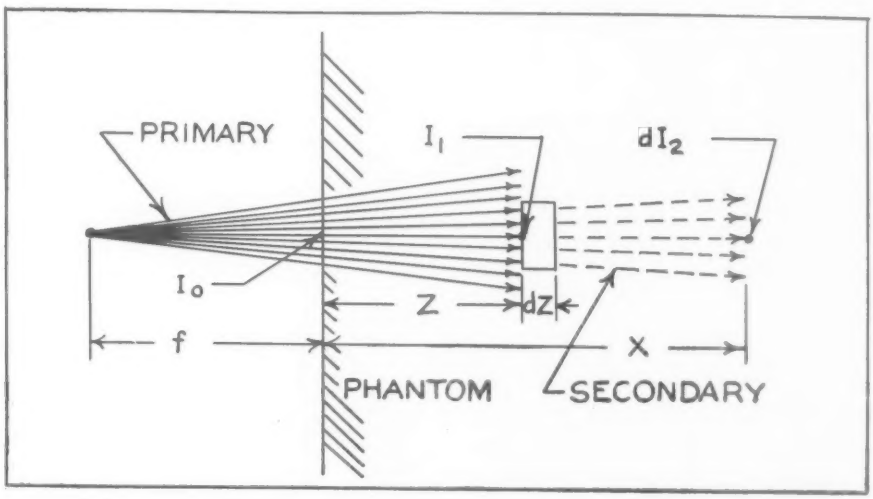

FIG. 7. Diagram to illustrate the absorption of high energy photons in a phantom.

surface of the phantom the flux density of these photons will be given by

$$I_1 = I_0 e - \mu_1 z \left(\frac{f}{f+z}\right)^2 \qquad (1)$$

where f is the focal skin distance and μ_1 is the absorption coefficient for the primary radiation. Between z and $z+dz$ a number $\mu_1 I_1 dz$ will be absorbed and will produce a proportional number of secondaries con-

sisting largely of Compton electrons and electron pairs. If only Compton electrons were produced the number of secondaries would be equal to the number of primary quanta involved If only electron pairs were produced the number of secondaries would be doubled. In any case for a given type of radiation the number of secondaries produced will be equal to $K_1\mu_1 I_1 dz$ where K_1 is a constant. If we assume that all the secondaries from dz are projected in the forward direction the flux density of secondaries at a depth x will be given by

$$dI_2 = K_1\mu_1 I_1 e^{-\mu_2(x-z)}\left[\frac{f+z}{f+x}\right]^2 dz$$

$$= K_1\mu_1 I_0\left[\frac{f}{f+x}\right]^2 e^{-\mu_1 z+\mu_2 z-\mu_2 x}dz \qquad (2)$$

where μ_2 is the absorption coefficient for the secondaries. In reaching a depth x the secondaries must pass through $(x-z)$ of absorbing material and so will be reduced by the factor $e^{-\mu_2(x-z)}$.* Since we have assumed that they are all projected in the forward direction their flux density will be reduced by the factor $\left[\frac{f+z}{f+x}\right]^2$ due to inverse square law (see Fig. 7). The density of secondaries reaching a depth x will be found by integrating expression (2) from $z=0$ to $z=x$ as follows:

$$I = \int_{z=0}^{z=x} dI_2$$

$$= \frac{K_1\mu_1 I_0}{\mu_2-\mu_1}\cdot\left(\frac{f}{f+x}\right)^2\left[e^{-\mu_1 x}-e^{-\mu_2 x}\right]. \qquad (3)$$

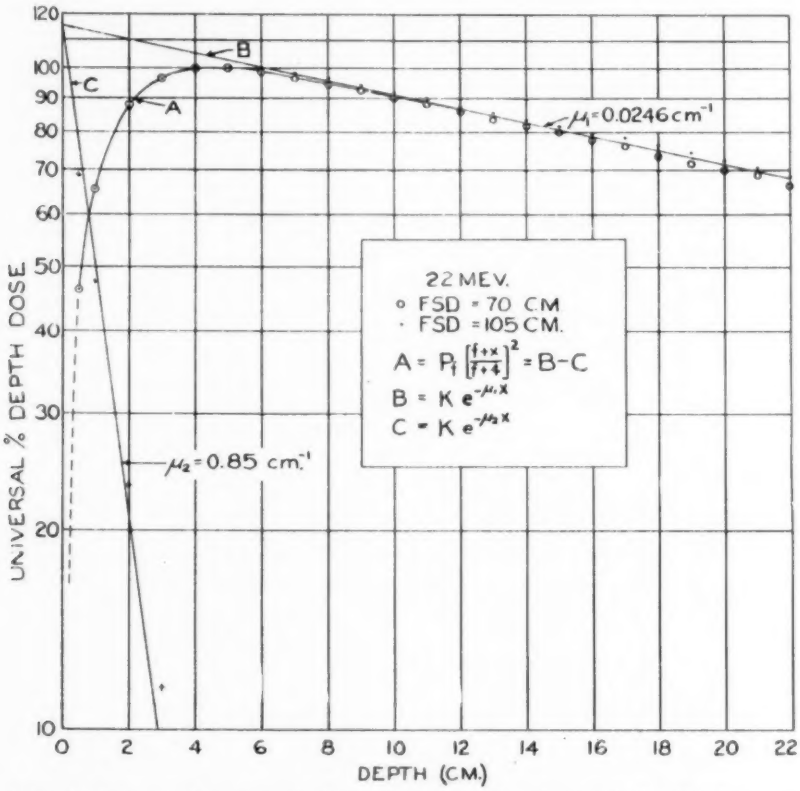

FIG. 8. Universal depth dose graph and analysis to show that the observed distribution actually can be obtained from the difference between two exponentials (22 mev. betatron radiation using a copper compensating filter).

* The assumption of an exponential law of absorption for the secondaries is admittedly open to question because of the complicated nature of the phenomenon but appears to be justified by the results.

The ionization density shown in Figure 6 is plotted as a percentage of the maximum which occurs at 4.0 cm. depth. Hence the percentage depth dose for any depth x is given by

$$P_f = 100\,\frac{I_x}{I_4} = 100\,\frac{(f+4)^2}{(f+x)^2}\,\frac{[e^{-\mu_1 x}-e^{-\mu_2 x}]}{[e^{-4\mu_1}-e^{-4\mu_2}]} = K\left(\frac{f+4}{f+x}\right)^2[e^{-\mu_1 x}-e^{-\mu_2 x}]. \quad (4)$$

In this expression the quantity $\left[\frac{f+4}{f+x}\right]^2$ gives the reduction in depth dose due to the inverse square law and the second term $[e^{-\mu_1 x}-e^{-\mu_2 x}]$ gives the dependence of depth dose on absorption. This dependence is rather complicated for small values of x when equilibrium is being established between the primaries and secondaries. For large values of x, $e^{-\mu_2 x}$ becomes negligible and we obtain a logarithmic type of absorption. For $x=4$, the above expression for P_f reduces to 100.

To test whether the actual data can be expressed by equation (4) the effect of inverse square law reduction was removed by calculating for each value of P_{70} and P_{105} in Table 1 the quantity $A = P_f\left[\frac{f+x}{f+4}\right]^2$.

It was found that for any given depth the two values for A agreed with one another to better than 3 per cent on the average. Hence only the average values for A are given in Table 1 under the heading of Universal Depth Dose. These values are the depth doses which would be obtained if there were no reduction due to inverse square law. The two values for A have been plotted in Figure 8 and it will be seen that both sets of points do lie along one curve which has the general form of the difference between two exponentials.

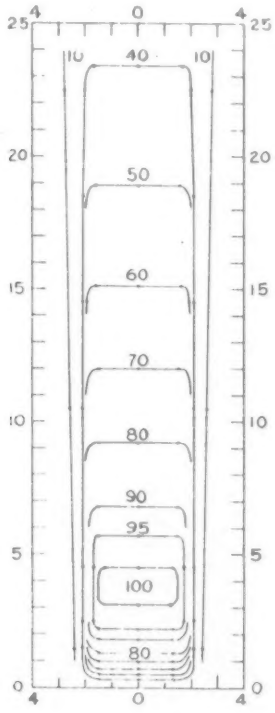

Fig. 9. Isodose distributions for a 4 cm. circular field at a focal skin distance of 70 cm. using radiation from a 22 mev. betatron with a copper compensating filter.

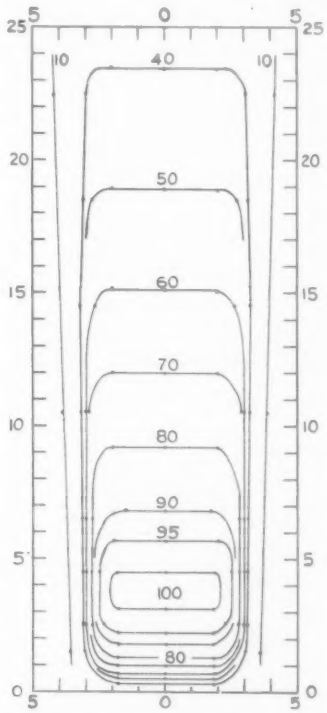

Fig. 10. Isodose distributions for a 6 cm. circular field at a focal skin distance of 70 cm. using radiation from a 22 mev. betatron with a copper compensating filter.

The graph in Figure 8 was analyzed by the standard methods yielding the two exponential curves B and C indicated in Figure 8. From the slope of lines B and C the absolute values for μ_1 and μ_2 were found to be 0.0246 cm.$^{-1}$ and 0.85 cm.$^{-1}$. The value of μ_1 is determined to within about 3 per cent while the value of μ_2 can be obtained with an accuracy of about 10 per cent. The value of μ_1 can be calculated from the known Klien-Nishina coefficients and the known cross section for pair production for water. The values for the total linear absorption coefficient are 0.051 cm.$^{-1}$ at 2 mev., 0.034 cm.$^{-1}$ at 4 mev., 0.023 cm.$^{-1}$ at 8 mev., 0.0185 cm.$^{-1}$ at 15 mev. and 0.016 cm.$^{-1}$ at 30 mev. The experimental linear absorption coefficient 0.0246 cm.$^{-1}$ obtained above is not inconsistent with these values and would correspond to an "average" photon energy of 7.5 mev.

The universal depth dose values (A) in Table 1 can be used to calculate the percentage depth dose (P_f) for any depth and any focal skin distance since P_f is given by $P_f = \left[\frac{f+4}{f+x}\right]^2 \cdot A$. For example, the depth dose for $f = 150$ cm. and $x = 10$ cm. would be $(91)\left[\frac{154}{160}\right]^2 = 84.3$ per cent.

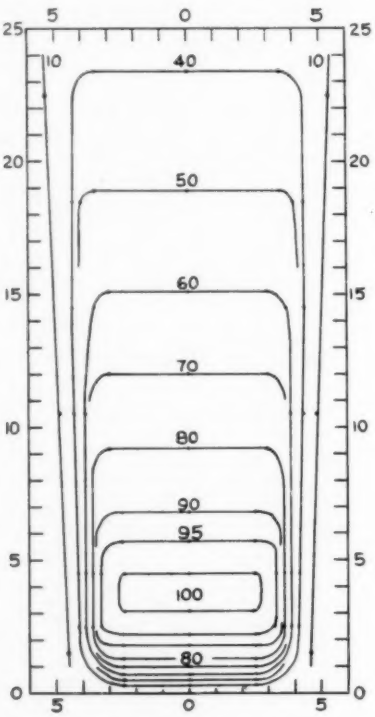

FIG. 11. Isodose distributions for an 8 cm. circular field at a focal skin distance of 70 cm. using radiation from a 22 mev. betatron with a copper compensating filter.

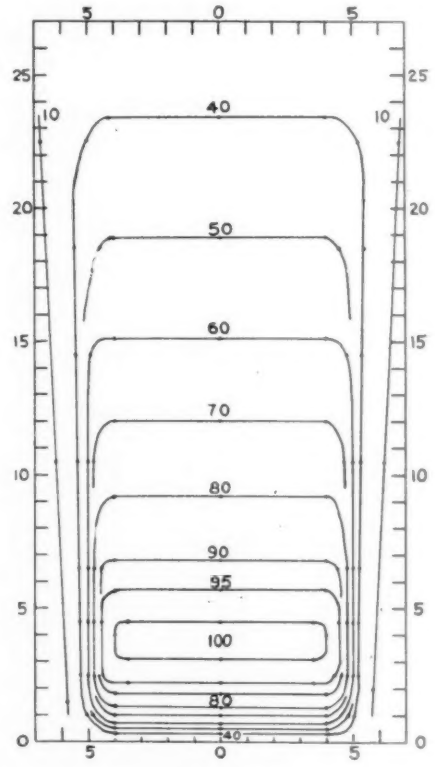

FIG. 12. Isodose distributions for a 10 cm. circular field at a focal skin distance of 70 cm. using radiation from a 22 mev. betatron with a copper compensating filter.

ISODOSE DISTRIBUTION

Isodose distributions for circular fields of diameter 4, 6, 8 and 10 cm. at 70 cm. focal skin distance and 6, 9, 12 and 15 cm. at 105 cm. are shown in Figures 9 to 16. These were obtained by making measurements of the radiation intensity at lattice points 1 cm. apart in a plane through the axis of the beam. Points of equal dose were connected to give the results indicated. The

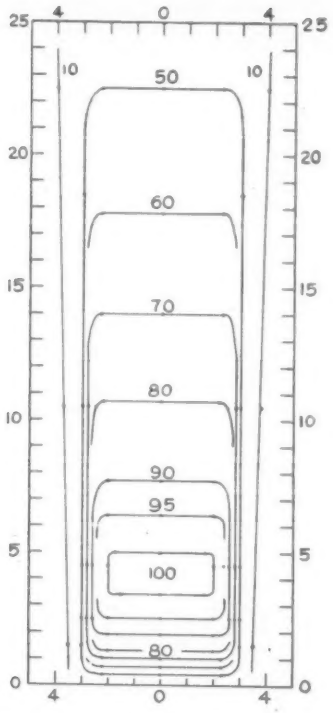

FIG. 13. Isodose distributions for a 6 cm. circular field at a focal skin distance of 105 cm. using radiation from a 22 mev. betatron with a copper compensating filter.

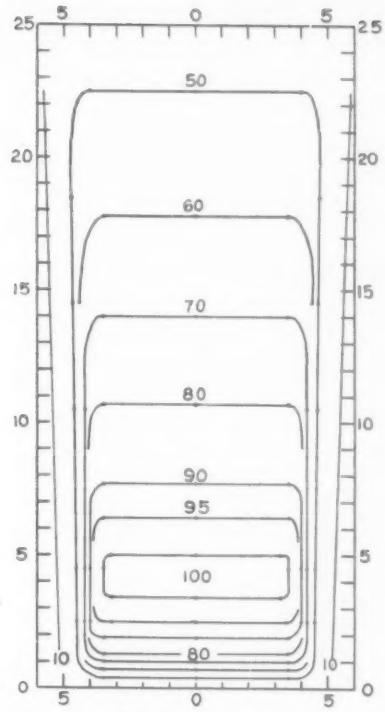

FIG. 14. Isodose distributions for a 9 cm. circular field at a focal skin distance of 105 cm. using radiation from a 22 mev. betatron with a copper compensating filter.

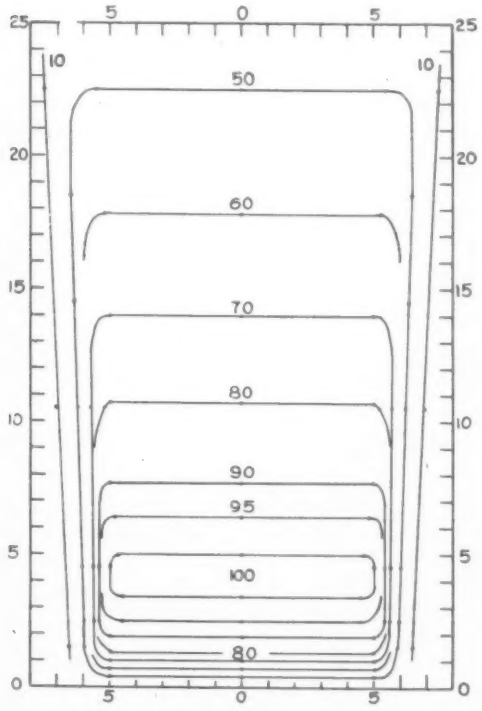

FIG. 15. Isodose distributions for a 12 cm. circular field at a focal skin distance of 105 cm. using radiation from a 22 mev. betatron with a copper compensating filter.

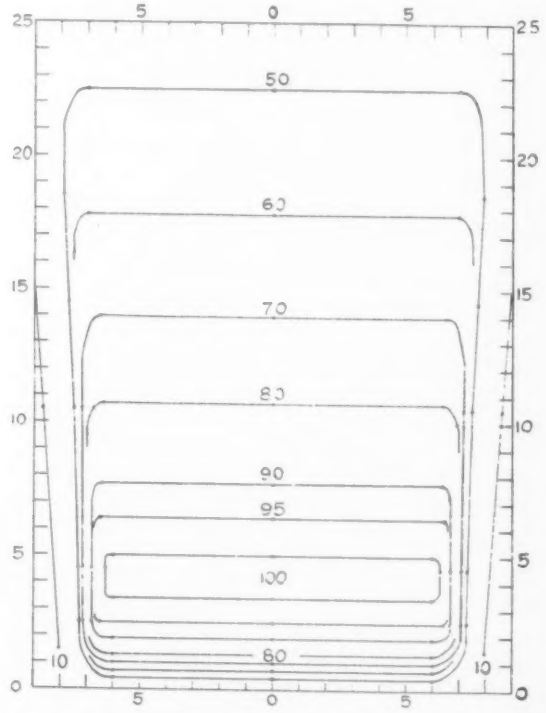

FIG. 16. Isodose distributions for a 15 cm. circular field at a focal skin distance of 105 cm. using radiation from a 22 mev. betatron with a copper compensating filter.

ionization chamber had a diameter of 6.0 mm. so that the radiation at a "point" could not be measured. This means the intensity of radiation was averaged over 6.0 mm. distance so that the contours are in error slightly in the regions where the radiation intensity changes rapidly. This means the regions of high dosage rate extend to slightly greater widths than are indicated in Figures 9 to 16. Examination of Figures 13 and 16 will indicate that if Figure 16 is selected the isodose distribution for a 6 cm. circular field (Fig. 13) can be obtained from the 15 cm. circular field by merely selecting the central 6 cm. from the larger distribution. This simplicity results from the fact that the depth dose is independent of area. It also means that the distributions in two planes at right angles for a 10 by 4 rectangular field are represented by Figures 9 and 12. It is hoped that these distributions will prove of value in formulating therapy treatments using the betatron.

SUMMARY

The roentgen-ray beam from a 22 mev. betatron has been carefully collimated and the intensity in the center of the beam reduced by the use of a copper compensating filter in such a way as to give a beam of uniform intensity over its cross section. A variety of different sized radiation fields were made available by the use of tapered lead plugs. Depth dose data were obtained in a water phantom using a small ionization chamber connected to a D.C. amplifier. It is found that the depth dose does not depend on the area of the field. A mathematical analysis of the depth dose curves indicates that they can be expressed as the difference between two exponentials. Isodore distributions for eight fields are presented.

The authors wish to acknowledge the support of the National Cancer Institute of Canada, the National Research Council of Canada and the Saskatchewan branch of the Canadian Cancer Society. They also wish to thank Dr. T. A. Watson, Director of the Saskatoon Cancer Clinic for his many helpful suggestions concerning roentgen-ray distributions. Thanks are due to Mr. S. B. Mauchel, betratron technician, for his assistance in taking readings, and to Mr. A. H. Cox of the instrument shop of the Physics Department for his careful work.

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Vol. 62, No. 2

THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

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Publisher: CHARLES C THOMAS, 301–327 East Lawrence Avenue, Springfield, Illinois.

Issued Monthly. Subscription $10.00 per year, $11.00 in Canada and $12.00 in foreign countries. Advertising rates submitted on application: Editorial office, 110 Professional Building, Detroit, Mich., Office of publication 301–327 East Lawrence Avenue, Springfield, Ill. Information of interest to all readers will be found on page iv.

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Thirty-second Annual Meeting: 1950, to be announced.

EDITORIALS

SIXTH INTERNATIONAL CONGRESS OF RADIOLOGY

I WELCOME the Editor's invitation to contribute this Editorial because of the chance it gives me of writing about the forthcoming Sixth International Congress of Radiology in London.

All going well, there will be a few readers of this JOURNAL who have not received a preliminary Programme of the Congress as this was mailed to Radiologists, Radiobiologists and Physicists in all those countries which have appointed Delegations. I want to use this opportunity of writing in an American journal to emphasize that a very warm invitation is extended to all readers to plan to come to London in July, 1950.

We have all of us in our own countries numerous Society Conferences and Annual Meetings; even wider groups foregather, too, from time to time such as the Pan-American Congress of this year and the excellent French-speaking Countries' Radiological Congress held in Geneva a short time ago. But the International Congress has always been something much more than these, and the British Committee has every intention that it will be so again. Scientifically perhaps the subjects would appear much the same as those on any other radiological programme at the present time. Indeed, it is extraordinary how little ground one can cover in a mere week, so extensive are the ramifications of Radiology today. But the speakers now bring opinions from the whole world to the rostrum and what is said on any subject should really be, for the moment at least, the last word on that subject.

But the scientific meetings per se are not the only important aspect of such gatherings. Indeed, I often wonder whether they are not merely the excuse—a sort of skeleton on which we hang the real flesh and blood of the Congress, the clash of minds and of ideas in private friendly intercourse. For this our International Congresses provide an opportunity on a world-wide scale. There one meets and talks to the leaders of every branch of Radiology. There one contacts like minds from all corners of the world and few should fail to find a kindred spirit, however eclectic their interest or hobby.

One thing I can promise those who come—the British Committee is very conscious of the value of informal discussion, the personal chat or gossip over coffee or the glass of beer, at dinner or at a reception, and they intend to provide adequate opportunity. But such opportunity will only serve its real function if people come determined to meet and to talk to their colleagues from countries other than their own; and, in fact, to seek them out if they are known to have worked in fields of Radiology in which the visitor is also interested. That is what the portrait catalogue is for. In addition, a ladies' programme for the wives and associate members, which looks as if it would be most attractive, has been drawn up—a programme so much more aptly described in French "programme de distraction."

I want, however, to use this Editorial to make another kind of proposal to American and Canadian Radiological Societies, Hospitals, Foundations, Governing Bodies. Travel to Europe is still a substantial expense. The leaders of Radiology, the Heads of Departments, the "high brass" of our specialty ought by now to be wealthy—or influential—enough to look after them-

selves. But what of the younger generation? For many of these, including the best intellects and the best original workers of today, the cost is heavy if self-borne entirely, and from what they learn they will bring back much of vital use to their home teams. Any hospital of any standing should be represented at such a Congress, but let them send one of their best younger men—men who would establish with their colleagues from other countries fruitful and lasting acquaintanceship founded on mutual respect. Indeed, I would like this Editorial to be taken as a form of application to governing bodies of such hospitals. Bodies such as A.E.C. and numerous cancer foundations and associations stand also to gain infinitely by being well represented at every relevant section of the Congress. Surely to send representatives from each of their institutions or laboratories is but enlightened self-interest. So can I plead with those in influential positions to get their Committees or Boards to offer Congress fellowships to well chosen members of their staffs?

One last proposal. How about the radiological societies themselves each offering five to ten—and the smaller societies appropriately less—Congress assistance grants of say $500 to $750 to open competition for the under forties of their membership. Perhaps in the interests of international good-will they might also offer one or two as a gesture to societies of some of the countries still very truthfully described as "war-impoverished."

But even if you are not wealthy and none of these ideas bear fruit in your direction my advice to all who mean to make their mark in radiology is to come anyway. Many before you have saved their pennies and travelled by old tramp steamers and now look back on Stockholm, Zurich or Paris as highlights of their radiological experience. Moreover, from the younger generation of today will come the leaders of future Congresses—and now is the time to start an association which should prove rich in experience and friendship in the years to come.

Ralston Paterson
President
Sixth International
Congress of Radiology

J. ERNEST GENDREAU
1880–1949

DR. J. ERNEST GENDREAU, a French-Canadian scientist of international reputation, especially interested in radiology and cancerology, and a member of the American Radium Society, died in Montreal, June 5, 1949, at the age of sixty-nine years, after a long illness.

He was the founder of the Institute of Radium of Montreal, and was one of the pioneers in physical sciences, and particularly in radiology, in Montreal. He held the Chair of Physics of the Faculty of Sciences of the University of Montreal until four years ago when he reached the age of retirement, at which time he was made professor emeritus. Two years ago he resigned from the direction of the Institute of Radium because of his poor health. His clinical studies were pursued in Canada and supplemented by periods of study in various European universities. He held the titles of Doctor of Medicine, Doctor of Philosophy, and Licentiate in Sciences, all obtained in Paris. He was formerly assistant to Drs. Jaugeas and Béclère in radiology; Madame Curie, Langevin and Regaud in radioactivity. After ten years of study in European universities he returned to Montreal where he founded the Institute of Radium.

James T. Case, M.D.

LUDWIG HALBERSTAEDTER
1877–1949

WHILE on a visit to the United States Dr. Halberstaedter died suddenly on April 21, 1949, at the age of seventy-two. He was born in Beuthen, Silesia, in 1877 and studied medicine at Breslau. He was one of the pioneer radiologists, becoming interested in radiations and publishing papers on light therapy and on roentgen rays in 1904 and 1905. After World War I he became Director of the Radiotherapy Section of the Berlin Cancer Institute. Dr. Halberstaedter was the first to recognize the specific action of roentgen rays on the ovaries and this recognition formed the basis for a great part of radiotherapy in gynecology. He also did important work on the treatment of skin cancer with radium molds. As early as 1932 he began work on million volt roentgen rays and cathode rays but in 1933 he was requested to resign his hospital appointment in Berlin and he considered it wise to leave Germany and went with his wife to Palestine. Here he took charge of radiotherapy at the Hadassah Hospital in Jerusalem and continued his research work at the Hebrew University, publishing many papers. At the time of his death he was Professor of Radiology at the Hebrew University and Chief of the Cancer Laboratories in Palestine.

Dr. Halberstaedter was well known personally to many American radiologists and he was a great scientist and friend.

Ira I. Kaplan, M.D.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: Netherland Plaza Hotel, Cincinnati, Ohio, Oct. 4–7, 1949.

American Radium Society
Secretary, Dr. H. F. Hare, 605 Commonwealth Ave., Boston, Mass. Annual meeting: 1950, to be announced.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: Cleveland Auditorium and Statler Hotel, Cleveland, Ohio, Dec. 4–9, 1949.

American College of Radiology
Executive Secretary, William C. Stronach, 20 N. Wacker Drive, Chicago 6. Annual meeting: 1950, to be announced.

Section on Radiology, American Medical Association
Secretary, Dr. Paul C. Hodges, 950 E. 59th St., Chicago, Ill. Annual Meeting: 1950, to be announced.

Alabama Radiological Society
Secretary, Dr. W. W. Anderson, Tuscaloosa, Ala. Meets time and place Alabama State Medical Association.

Arizona Association of Pathologists and Radiologists
Secretary, Dr. R. Lee Foster, 507 Professional Bldg., Phoenix, Ariz. Two regular meetings a year. The annual meeting at time and place of State Medical Association and interim meeting six months later.

Arkansas Radiological Society
Secretary, Dr. Fred Hames, 511 National Bldg., Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

Atlanta Radiological Society
Secretary, D. W. W. Bryan, 490 Peachtree St., N.E., Atlanta, Ga. Meets monthly, except during three summer months, on second Friday evening.

Brooklyn Roentgen Ray Society
Secretary, Dr. Joseph Daversa, 603 Fourth Ave., Brooklyn, N. Y. Meets monthly fourth Tuesday, Oct. to April.

Buffalo Radiological Society
Secretary, Dr. Mario C. Gian, 610 Niagara St., Buffalo, N. Y. Meets second Monday evening each month, October to May inclusive.

Central New York Roentgen Ray Society
Secretary, Dr. Dwight V. Needham, 608 E. Genesee St., Syracuse N. Y. Meets January, May, November.

Central Ohio Radiological Society
Secretary, Dr. Paul D. Meyer, Grant Hospital, Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, December, February, April, and June at Seneca Hotel, Columbus, Ohio.

Chicago Roentgen Society
Secretary, Dr. John H. Gilmore, 720 N. Michigan Ave., Chicago 11, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

Cincinnati Radiological Society
Secretary, Dr. Eugene L. Saenger, 735 Doctors Bldg., Cincinnati 2, Ohio. Meets last Monday of each month, September to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. J. R. Hannan, Cleveland Clinic, Cleveland 6, Ohio. Meetings at 6:30 P.M. on fourth Monday of each month from October to April.

Colorado Radiological Society
Secretary, Dr. Mark S. Donovan, 306 Majestic Bldg., Denver 2, Colo. Meets third Friday of each month at Department of Radiology, Colorado School of Medicine.

Connecticut Valley Radiologic Society
Secretary, Dr. E. W. Godfrey, 1676 Boulevard, West Hartford, Conn. Meets second Friday Oct. and April.

Dallas-Fort Worth Roentgen Study Club
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meets in Dallas on odd months and in Fort Worth on even months, on third Monday, 7:30 P.M.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. W. G. Belanger, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

East Bay Roentgen Society
Secretary, Dr. Dan Tucker, 434-30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

Florida Radiological Society
Secretary, Dr. F. K. Hurt, Riverside Hospital, Jacksonville, Fla. Meets twice annually, in the spring with the annual State Society meeting, and in the fall.

Georgia Radiological Society
Secretary, Dr. Robert Drane, DeRenne Apartments, Savannah, Ga. Meets in mid-winter and at annual meeting of Medical Association of Georgia in the spring.

Houston X-ray Club
Secretary, Dr. Curtis H. Burge, 3020 San Jacinto St. Houston 4, Texas. Meets fourth Monday each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Illinois Radiological Society
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. William M. Loehr, 712 Hume-Mansur Bldg., Indianapolis 4. Meets second Sunday in May.

Iowa X-Ray Club
Secretary, Dr. Arthur W. Erskine, 326 Higley Bldg., Cedar Rapids, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. Special meetings by announcement.

Kansas Radiological Society
Secretary, Dr. Anthony F. Rossitto, Wichita Hospital, Wichita, Kan. Meets annually with State Medical Society.

Kentucky Radiological Society
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

Kings County Radiological Society
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Los Angeles Radiological Society
Secretary, Dr. Wybren Hiemstra, 1414 S. Hope St., Los Angeles 15, Calif. Meets second Wednesday each month at Los Angeles County Medical Assn. Building.

Louisiana Radiological Society
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

Louisville Radiological Society
Secretary, Dr. E. L. Pirkey, Louisville General Hospital, Louisville 2, Ky. Meets monthly on second Friday at Louisville General Hospital.

* Secretaries of societies are requested to send timely information promptly to the Editor.

Michigan Association of Roentgenologists
Secretary, Dr. R. D. McDuff, 220 Genesee Bank Bldg., Flint 3, Mich.

Milwaukee Roentgen Ray Society
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. Chauncey N. Borman, 802 Medical Arts Bldg., Minneapolis 2, Minn. Two meetings yearly, one at time of Minnesota State Medical Association the other in the fall.

Nebraska Radiological Society
Secretary, Dr. Ralph C. Moore, Nebraska Methodist Hospital, Omaha 3, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. George Levene, Massachusetts Memorial Hospitals, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

New Hampshire Roentgen Ray Society
Secretary, Dr. A. C. Johnston, Elliott Community Hospital, Keene, N. H. Meets four to six times yearly.

New York Roentgen Society
Secretary, Dr. F. H. Ghiselin, 111 East 76th St., New York 21, N. Y. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

North Carolina Radiological Society
Secretary, Dr. J. E. Hemphill, 1420 E. Fifth St., Charlotte 4, N. C. Meets in May and October.

North Dakota Radiological Society
Secretary, Dr. C. O. Heilman, 807 Broadway, Fargo. Meetings held by announcement.

Northern California Radiological Club
Secretary, Dr. C. E. Grayson, Medico-Dental Bldg., Sacramento 14, Calif. Meets at dinner last Monday, every second month, except June, July and August.

Ohio State Radiological Society
Secretary, Dr. E. C. Elsey, 927 Carew Tower, Cincinnati 2, Ohio.

Oklahoma State Radiological Society
Secretary, Dr. W. E. Brown, Tulsa, Okla. Three regular meetings annually.

Oregon Radiological Society
Secretary, Dr. Boyd Isenhart, 214 Medical Dental Bldg., Portland 5, Oregon. Meets monthly 2nd Wednesday, 8:00 P.M., Library of University of Oregon Medical School.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets first Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. S. J. Hawley, 1320 Madison St., Seattle 4, Wash. Meets annually in May.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. J. M. Converse, 416 Pine St., Williamsport. Annual Meeting: May 20 and 21, 1949, Bedford Springs Hotel, Bedford, Pa.

Philadelphia Roentgen Ray Society
Secretary, Dr. Arthur Finkelstein, Graduate Hospital, 19th and Lombard St. Meets first Thursday each month October to May, at 8:00 P.M., in Thomson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. R. P. Meader, 4002 Jenkins Arcade Pittsburgh 22, Pa. Meets 6:30 P.M. at Webster Hall Hotel on second Wednesday each month, October to May inclusive.

Queens Roentgen Ray Society
Secretary, Dr. J. E. Goldstein, 88-29 163rd St., Jamaica 3, N. Y. Meets fourth Monday of each month except during the summer.

Radiological Section, Baltimore Medical Society
Secretary, Dr. Harry A. Miller, 2452 Eutaw Place, Baltimore. Meets third Tuesday each month, September to May.

Radiological Section, Connecticut Medical Society
Secretary, Dr. Fred Zaff, 135 Whitney Ave., New Haven, Conn. Meets bimonthly on second Wednesday.

Radiological Section, District of Columbia Medical Society
Secretary, Dr. A. A. J. Den, 1801 K St., N. W., Washington, D. C. Meets Medical Society Auditorium, third Thursday, January, March, May, October at 8:00 P.M.

Radiological Section, Southern Medical Association
Secretary, Dr. Roy G. Giles, Temple, Texas.

Radiological Society of New Jersey
Secretary, Dr. Raphael Pomeranz, 31 Lincoln Park, Newark, N. J. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Ralph E. Alexander, 101 Medical Arts Bldg. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. Maurice D. Frazer, 1037 Stuart Bldg., Lincoln, Nebr. Meets Shirley-Savoy Hotel, Denver, Colo. August 18, 19, 20, 1949.

St. Louis Society of Radiologists
Secretary, Dr. C. J. Nolan, 737 University Club Bldg., St. Louis 3, Mo. Meets fourth Wednesday each month, except June, July, August, and September.

San Diego Roentgen Society
Secretary, Dr. R. F. Niehaus, 1831 Fourth Ave., San Diego, Calif. Meets monthly, first Wednesday at dinner.

Section on Radiology, California Medical Association
Secretary, Dr. D. R. MacColl, 2007 Wilshire Blvd., Los Angeles 5, Calif.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. Harold L. Shinall, St. Joseph's Hospital, Bloomington, Ill.

Shreveport Radiological Club
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon. 650 Fifth Ave., Fort Worth 4, Texas. Next meeting, Dallas, Texas, February 3 and 4, 1950.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets first and third Thursdays 4:00 to 5:00 P.M., September to May inclusive. Room 203, Service Memorial Institute, 426 N. Charter St., Madison.

Utah State Radiological Society
Secretary, Dr. Angus K. Wilson, 343 S. Main St., Salt Lake City 1, Utah. Meets third Wednesday in September, November, January, March and May.

Virginia Radiological Society
Secretary, Dr. P. B. Parsons, Norfolk General Hospital, Norfolk, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Homer V. Hartzell, 310 Stimson Bldg., Seattle 1, Wash. Meets fourth Monday each month, October through May, College Club, Seattle.

X-Ray Study Club of San Francisco
Secretary, Dr. W. F. Reynolds, University of California Hospital, San Francisco. Meets from January to July, 1949, at Lane Hall, Stanford University Hospital, and from July to December 1949, at San Francisco Hospital.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

Mexico

Sociedad Mexicana de Radiologia y Fisioterapia
General Secretary, Dr. D. P. Cossio, Marsella No. 11, Mexico, D. F. Meets first Monday of each month.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Ordinary meeting, on the Thursday preceding the third Friday, October to May at 8:15 p.m.
Medical Members' meeting, on third Friday in each month at 5:00 p.m., 32 Welbeck St., London, W 1.

Faculty of Radiologists
Honorary Secretary, Dr. J. F. Bromley, 45, Lincoln's Inn Fields, London, W.C.2, England.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 p.m. at the Royal Society of Medicine, 1 Wimpole St., London.

Canadian Association of Radiologists
Honorary Secretary, Dr. E. M. Crawford, 1535 Sherbrooke St., West, Montreal 26, Que. Meetings January and June.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Francaise d'Electrologie et de Radiologie Médicales
Secretary, Dr. Origéne Dufresne, 4120 Ontario St., East, Montreal, P. Q.

Australian and New Zealand Association of Radiologists
Honorary Secretary, Dr. Alan R. Colwell, 135 Macquarie St., Sydney, N.S.W.
Honorary Secretaries, State Branches:
New South Wales, Dr. E. W. Frecker, 135 Macquarie St., Sydney.
Victoria, Dr. T. I. Tyrer, 3 Lockerbie Court, East St. Kilda.
Queensland, Dr. J. Adam, 131 Wickham Terrace, Brisbane.
South Australia, Dr. B. C. Smeaton, 178 North Terrace, Adelaide.
Western Australia, Dr. A. M. Nelson, 179-B St. Georges Terrace, Perth.
New Zealand, Dr. E. G. Lynch, 12 Bolton St., Wellington.

South America

Sociedad Argentina de Radiologia
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Sociedade Brasileira de Radiologia Medica
Secretary, Dr. Nicola Caminha, Av. Mem de Sa, Rio de Janeiro, Brazil. Meets monthly, except during January, February and March.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Andrelino Amaral, Av. Brigadeiro Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Tuesday at 9 p.m. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Peruana de Radiologia
Secretary, Dr. Julio Bedoya Paredes, Apartado, 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta, 218, Lima.

Sociedad de Radiologica, Cancerologia y Fisica Medica del Uruguay
Secretary, Dr. Arias Bellini.

Continental Europe

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 111 Avenue des Alliés, Louvain, Belgium. Meets monthly, second Sunday at Maison des Médecins, Brussels.

Ceskoslovenská společnost pro röntgenologii a radiologii v Praze
Secretary, Dr. Roman Bláha, Praha XII, stát. nemocnice, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, 59 Nowogrodzka St., Warsaw, Poland. Next meeting, Krakow, June 2 and 3, 1949.

Gdansk Section, Polish Society of Radiology
Secretary, Dr. A. Smigielska, Akademia Lekarska, Gdansk. Meets monthly last Sunday at 10.30, X-Ray Dept., Akademia Gdansk.

Warsaw Section, Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, 59 Nowogrodzka St., Warsaw, Poland. Meets monthly.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banual Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad.
USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 p.m.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Schweizerische Röntgen-Gesellschaft (Société Suisse de Radiologie)
President, Dr. H. E. Walther, Gloriastr. 14, Zürich, Switzerland.

Societa Italiana di Radiologia Medica
Secretary, Prof. Mario Ponzio, Ospedale Mauriziano Torino, Italy. Meets biannually.

HOTEL RESERVATIONS AMERICAN ROENTGEN RAY SOCIETY

The July issue of the JOURNAL carries the Preliminary Program and the Instruction Courses for the meeting of the American Roentgen Ray Society to be held at the Netherland Plaza Hotel, Cincinnati, Ohio, October 4–7, 1949. In the Editorial in that issue attention was called to the importance of making hotel reservations at an early date. This is again emphasized. While the Netherland Plaza and the Terrace Plaza have been most generous in their allotment of rooms for those attending the meeting, it will still be necessary to use other Cincinnati hotels in close proximity to the meeting place. An adequate guarantee of rooms has been made by all these hotels in cooperation with the Convention Bureau but these rooms will be held for the Society only up to thirty days in advance of the meeting.

It is therefore urged that all those who contemplate attending the meeting in Cincinnati make their reservations at once. It is always easier, if at the last minute something unforeseen prevents attendance at the meeting, to cancel a reservation than it is to secure a room at a late date.

Cooperation in this respect by everyone anticipating attending the meeting will be appreciated by the hotels, the Convention Bureau and the Society. All reservations should be made through the Netherland Plaza Hotel, Cincinnati 2, Ohio.

ATLANTA RADIOLOGICAL SOCIETY

A called meeting of the radiologists of the city of Atlanta was held on the evening of March 11, 1949. At that meeting it was decided that a society of radiologists should be formed in the city of Atlanta, this society to be named the Atlanta Radiological Society. The following officers were elected: *President*, Dr. J. J. Clark; *Vice-President*, Dr. Charles A. Priviteri; *Secretary-Treasurer*, Dr. William W. Bryan. Meetings will be held monthly, except during the three summer months, on the second Friday evening.

KINGS COUNTY RADIOLOGICAL SOCIETY

At a recent meeting, the name of the Long Island Radiological Society was changed to the Kings County Radiological Society. The following officers were elected for the ensuing year: *President*, Dr. H. G. Koiransky; *Vice-President*, Dr. I. Silverstein; *Secretary*, Dr. Marcus Wiener (re-elected); *Treasurer*, Dr. M. H. Goldenberg.

NEW YORK ROENTGEN SOCIETY

At a recent meeting of the New York Roentgen Society the following officers were elected for the coming year: *President*, Dr. William Snow; *Vice-President*, Dr. Robert P. Ball; *Secretary*, Dr. F. H. Ghiselin; *Treasurer*, Dr. Harold L. Temple.

ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

Department Editor: George M. Wyatt, M.D., 1835 Eye St., N.W., Washington 6, D. C.

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ROENTGEN DIAGNOSIS

HEAD

McRae, Donald L. Focal epilepsy; Correlation of the pathological and radiological findings. *Radiology*, April, 1948, *50*, 439–457.

The author discusses classification on anatomical basis, and changes seen on plain roentgen films of skull and by encephalography on 160 patients with focal epilepsy due to atrophic cerebral and meningocerebral lesions.

The most common atrophic lesion, meningocerebral cicatrix, was associated with localized bony abnormalities, namely, old fracture, old bone defect, craniocerebral erosion, or localized thinning and bulging of the bone in 66 per cent of cases. Reduction in volume of one lateral chamber of skull noted in half of the cases. The incidence of localized bone changes is six times greater than in any other type of lesion. Craniocerebral erosion seems to be a specific finding. Pneumograms demonstrated focal ventricular dilatation in 80 per cent of the patients with meningocerebral cicatrix.

The second largest group consists of simple atrophic lesions, namely, focal microgyria, cerebral cicatrix secondary to arterial or venous occlusions, and brain cysts secondary to cerebral hemorrhage. Localized bony abnormalities occurred in only 11 per cent of this group. Cranial hemiatrophy was present in about half of the cases. Focal ventricular dilatation was demonstrated in 38 per cent and subarachnoid cysts in 5 per cent of these patients.

The third group included blood vessel anomalies. Intracerebral calcification is a common finding. Occasionally changes were found suggesting a space-occupying lesion.

The demonstration of a focal atrophic lesion did not always localize the epileptogenic focus. —*E. E. Seedorf, M.D.*

Wyatt, J. P., and Goldenberg, H. Craniolacunia. *Arch. Path.*, May, 1948, *45*, 667–677.

This paper is a report of 6 cases of craniolacunia (Lückenschädel) found at autopsy in one year at the Toronto East General Hospital (representing about 10 per cent of their 62 stillborn and neonatal deaths) along with a brief review of the literature. In their 6 cases the authors found defects only in the membranous bones—the abnormalities being similar in all but extent and degree. The defects were primarily of the inner tables, sometimes both tables, and they were surrounded by interlacing bony ridges. The defects do not conform to the convolutions of the brain, nor to the centers of ossification. In all cases there were associated anomalies involving the central nervous system, the most frequent being spina bifida and meningocele, as has been previously reported. The authors do not consider increased intracranial pressure per se to be the vital etiological factor, leaning more to the view that it is simply an anomaly of skeletal development.—*D. D. Beiler, M.D., and C. L. Hinkel, M.D.*

Weber, G. Zur Diagnose und Behandlung der arteriovenosen Aneurysmen im Bereich der Grosshirnhemisphären. (The diagnosis and therapy of cerebral arteriovenous aneurysms.) *Schweiz. med. Wchnschr.*, July 3, 1948, *78*, 629–634.

Cerebral arteriography is regarded as particularly valuable in the diagnosis of cerebral arteriovenous aneurysms. In the 15 cases observed by the author, only in 4 could a systolic-diastolic murmur be heard over the temporal regions. None of his cases had any detectable calcifications roentgenologically. Encephalography revealed dilated ventricles in 5 and a deviation of one ventricle in 1 instance only. Cardiac enlargement or unilateral exophthalmos was not observed. The author's indications for cerebral arteriography are (1) history of subarachnoid hemorrhage, (2) unilateral jacksonian attacks without encephalographic findings, (3) combination of subarachnoid hemorrhage and jacksonian attacks.—*Arthur Grishman, M.D.*

Wolfe, H. R. I. Unexplained thrombosis of the internal carotid artery. *Lancet*, Oct. 9, 1948, *2*, 567–569.

A case of complete occlusion of the left internal carotid artery in a female, aged twenty-six, is presented. This is the only case of complete occlusion the author found in 35 cases described in the literature. Thirty-two previously described cases diagnosed by arteriography are discussed as to pathology, diagnosis and treatment. The angiogram of the present case is reproduced showing a cone-shaped termination of the thorotrast column just below the atlas.—*J. S. Summers, M.D.*

Jacoby, N. M., and Sagorin, L. Human toxoplasmosis in England. *Lancet*, Dec. 11, 1948, *2*, 926–928.

Although the presence of toxoplasma in animals was described in England as early as 1914, this is the first recorded case in a human in that country.

Only one case, that of a male infant four months of age, is presented. There is a photograph of the typical "scissors" position and a reproduction of the skull films showing calcification.

The author concludes that the disease may well be more common in England than is supposed. Cases may be misdiagnosed congenital chorioretinitis, encephalitis and chronic meningitis.—*J. S. Summers, M.D.*

Schulz, Milford D., and Heath, Parker. Lymphoma of the conjunctiva. *Radiology*, April, 1948, *50*, 500–505.

Authors present 14 lymphomas taken from 1,600 records of patients with lymphomas of all varieties. Five of them were female, and 9 male. In this series the disease was found in elderly patients, almost half of the group being more than seventy years of age and all but 3 over fifty years of age.

Cytologically, 11 of the 14 were lymphocytomas, two having predominantly reticulum cells and another had many lymphoblasts.

None of the 14 patients showed an abnormal blood picture at time of entry. Of the tumors localized to the conjunctiva, 4, or 50 per cent, were infiltrative and none showed evidence of recurrence or remote manifestations. Of the 6 with remote disease, all but 2 were invasive tumors, lymphosarcoma, and all promptly succumbed to their disease except one. This histopathologic character of lymphomatous disease seems to have no conclusive bearing on prognosis.

All tumors were treated with radium or roentgen radiation save two which were excised. Treatment factors, 200 kv., 20 cm. object target distance, 0.25 mm. copper filter. Daily dose of 200 to 300 r was given, directed to conjunctiva for a total of 600 to 900 r. No untoward effect noted upon eye.

None of the patients in whom the lymphoma was localized to the conjunctiva when first seen has had reappearance of disease after treatment. Six have been well two years or more; 2 with invasive tumors more than five years. In contrast, all of the patients whose conjunctival involvement was part of a generalized lymphoma were dead in less than two years or have uncontrolled disease. The eyes in none, however, show recurrence.—*Robert D. Moreton, M.D.*

NECK AND CHEST

Meyer, Alfred C., Dockerty, Malcolm B., and Harrington, Stuart W. Inflammatory carcinoma of the breast. *Surg., Gynec. & Obst.*, Oct., 1948, *87*, 417–424.

That certain malignant lesions of the breast may simulate inflammatory lesions clinically has long been a matter of common knowledge. Although clinically these lesions do not present the usual picture of carcinoma of the breast and often have been mistaken for inflammatory lesions, pathologically they represent a very virulent type of cancer.

The purpose of this study was to describe and classify this type of disease, to investigate its pathologic characteristics and to correlate its clinical manifestations with its pathologic characteristics.

Summary

In 74 (1 per cent) of approximately 7,000 cases of malignant lesion of the breast, the diagnosis was inflammatory carcinoma because of characteristic clinical features which simulated those of inflammation. The disease was found to occur in the same general age group as carcinoma of the breast and there was no particular correlation with pregnancy or lactation. The incidence of bilateral malignancy was two times that noted in comparable cases of the usual carcinoma of the breast. The left breast was affected primarily nearly twice as frequently as the right. On examination both breasts were large and often the involved one had become bigger than its mate. The skin over the breasts was red or edematous or both, and palpation frequently revealed diffuse infiltration rather than a localized tumor. The axillary nodes were involved in all cases.

Pathologically the lesions were diffuse, high grade adenocarcinomas which frequently involved the lymphatics and occasionally the blood vessels. Characteristically the cancer spread through the subepidermal lymphatics. There was no evidence of bacterial infection, and ulceration of the skin was extremely rare. The inflammatory appearance was apparently due to blockage of the lymphatics by cancer cells and the resultant vascular phenomena.

Prognosis for life is poor but 3 patients lived more than five years after operation and 1 was alive nine years after operation.

The common sites of metastasis were the thorax, bone, the opposite breast and the skin. Occurrence of metastasis to the skin was especially frequent, having been present in 55 per cent of the cases in which metastasis was known to have occurred.—*Mary Francis Vastine, M.D.*

Tice, George I., Dockerty, Malcolm B., and Harrington, Stuart W. Comedomastitis; clinical and pathologic study of data in 172 cases. *Surg., Gynec. & Obst.*, Nov., 1948, *87*, 525–540.

Comedomastitis is a disease of the breast characterized by dilatation of the lactiferous ducts which are distended with inspissated grumous material that may be expressed from the cut ends of the ducts much as comedones are expressed from ordinary blackheads. It is a disease of the large ducts in contrast to chronic cystic mastitis which is a disease of the acini and ductules. Marked clinical similarity to mammary malignancy may be present when comedomastitis results in an indurated area of the breast which is fixed to the skin and is as-

sociated with a retracted nipple. Even the pathologic picture may be confused with that of carcinoma unless the true nature of the lesion is appreciated.

Conclusions.

1. Comedomastitis is a disease characterized by stasis of secretion, dilatation of the ducts, and periductal mastitis.

2. The clinical features of the disease vary widely, but the disease may be suspected in the presence of a history of abnormality of lactation, nonpuerperal mastitis, discharge from the nipple or abnormality of the nipple. On examination the lesion may closely simulate carcinoma because of the indurated, fixed mass and retracted nipples.

3. Comedomastitis usually is associated with chronic cystic mastitis which may overshadow it clinically.

4. Neoplasia, although it occasionally occurs in the same breast as does comedomastitis, is probably not related to the latter etiologically. By simple obstruction of the mammary ducts such neoplastic changes may, at times, produce a focal comedomastitis.

5. Plasma cell mastitis is probably a form of comedomastitis.—*Mary Frances Vastine, M.D.*

Bradbury, F. C. S. Minimal requirements for mass radiography. *Lancet*, Aug. 21, 1948, *2*, 293.

There is a lower limit to the efficiency of mass roentgenography, below which it becomes uneconomical. The author suggests that this lower limit would be the point where mass roentgenography leaves undiscovered more tuberculosis than it discovers.

On this basis not less than 70 per cent of any group to be surveyed should be actually examined if the return is commensurate with the expense.

The figure 70 per cent was chosen because in a series of mass surveys the amount of tuberculosis detected was shown to be directly proportional to the percentage response from any given group. That means that of the 70 per cent who had films made, only 70 per cent of the expected incidence of tuberculosis will be detected. Hence, if 100 per cent examination gives all the detectable tuberculosis, a 70 per cent response will give only $(70)^2/100$, or 49 per cent, of all the detectable tuberculosis.

Therefore, not less than 70 per cent of any group to be surveyed must be examined if the venture is to be an economical success.—*J. S. Summers, M.D.*

Tattersall, W. H. Diagnostic fluoroscopy at a chest clinic. *Lancet*, Dec. 18, 1948, *2*, 974–975.

The author is a chest physician of Reading, England, who believes that fluoroscopy in skilled hands can gain a high degree of accuracy without danger to the patient or operator.

Because of the fluoroscope's advantages of cheapness and rapidity of use he offered a rapid fluoroscopy service. During the two years of operation of this service, 2,908 patients have been examined. Three hundred fourteen cases were selected for roentgenography and further study. Eighty-three (26 per cent) of these were found to have tuberculosis. This means an overall discovery of 3 per cent tuberculosis in 2,908 cases. Thirty-two (39 per cent) of the 83 had positive sputum. No false negative reports have been found.

The author believes that 20 people can be adequately examined in half an hour but stresses complete dark adaptation.—*J. S. Summers, M.D.*

Horst, H. G., Legler, H., and Buchtala, V. Beitrag zur Differentialdiagnose der multiplen Herdschatten der Lunge. (Contribution to the differential diagnosis of multiple pulmonary densities.) *Schweiz. med. Wchnschr.*, June 5, 1948, *78*, 543–544.

Two cases of transient pulmonary edema with hemoptosis are described. In the first case the patient fell into a river while working near it. He was admitted to the hospital in an unconscious condition. Since rales were heard a chest roentgenogram was taken which showed multiple lobular cherry-sized infiltrations, particularly in the perihilar region. There was rapid recovery with one recurrence.

The second patient was suffering from epilepsy, being admitted half an hour after an attack of pulmonary edema. The chest roentgenogram similarly showed numerous, confluent bronchopneumonic-like consolidations.

To think of pulmonary edema of central origin in drowning accidents or acute cerebral episodes is clinically important since often no adequate history can be obtained in such cases. —*Arthur Grishman, M.D.*

Fleischner, Felix G. The visible bronchial tree; a roentgen sign in pneumonic and other

pulmonary consolidations. *Radiology*, Feb., 1948, *50*, 184–189.

The air-filled bronchial tree, not roentgenographically visible in a normal chest, may become visible as a ramifying system of radiolucent strands within a radiopaque field in the presence of lung disease causing absence of normal parenchymal alveolar aeration. The author presents several cases illustrating the diagnostic usefulness of this "visible bronchial tree" sign. It is most frequently helpful in recognizing all types of minimal pneumonic consolidation, especially in infants and children, and in obscure retrocardiac, basal, or lower lobe location. Infarction, tumors, bronchiectasis, and atelectasis may also show this sign.

The author describes an experiment demonstrating the reason for the sign. Crossed rows of paraffin-filled and air-filled paper drinking straws were roentgenographed twice in an open, paraffin-coated, paper box, once containing air, and once filled with water. Only the paraffin-filled straws were visible on the roentgenogram of the box containing no water. Only the air-filled straws were visible, as black lines in a white field, on the roentgenogram of the box filled with water.

Absence of the sign does not rule out pneumonic consolidation, since failure to demonstrate the bronchi may be due to the bronchial air being displaced by secretion, edema fluid, or blood, or due to over- or under-exposure of the roentgenograms.—*Richard E. Lukes, M.D.*

Bulgrin, James G. Unusual Friedländer's bacillus pneumonia associated with septicemia. *Radiology*, April, 1948, *50*, 526–528.

The author presents a report of an unusual case of Friedländer's bacillus pneumonia associated with septicemia due to that organism and characterized by nodular pulmonary densities of a type usually seen with tuberculosis or metastatic neoplasm. Complete resolution was obtained by streptomycin therapy.

A review of the literature reveals no similar case due to Friedländer bacillus.

Due to the efficiency of antibiotic therapy, recognition of unusual pulmonary lesions associated with septicemia of any kind is important.—*Robert D. Moreton, M.D.*

Herbert, F. J. Pulmonary tuberculosis in the old. *Lancet*, Aug. 14, 1948, *2*, 247–249.

Pulmonary tuberculosis in the aged is not rare and it may be highly infective. The present study covers the years 1938–1946 at the Stobhill Hospital, Glasgow. Sixty-nine cases of tuberculosis were found in patients over sixty years of age. Sixty of these were men. The higher rate in men was attributed to the fact that the majority of these men lived in "model lodging houses" which necessitated their admission to the hospital for even minor nursing care. More women presumably lived in homes where minor nursing care could be provided.

Tubercle bacilli were found in the sputum in 34 of the 69 cases. In 30 of the 35 cases with negative sputum, the examinations were considered in retrospect to have been inadequate.

A positive family history was obtained in only 13 cases (22 per cent). A previous history of pleurisy or of treatment for tuberculosis was given in 20 cases out of 63 (32 per cent).

Roentgen reports were available in 57 cases (83 per cent). Fibroid disease was present in 29 cases (51 per cent of the 57 cases) and it was bilateral in 15 (26 per cent). Active (fibrocaseous) disease was found in 24 cases (42 per cent). This was bilateral in 8 (14 per cent). There were 4 unclassified cases.

The author concludes that more attention should be directed to the problem of pulmonary tuberculosis in the aged. It has an insidious onset with symptoms commonly ascribed to old age. It is often an active process with a high proportion of sputum positive cases, dangerous to younger people around them.—*J. S. Summers, M.D.*

Medlar, E. M., Spain, David M., and Holliday, Robert W. Disregarded seedbed of the tubercle bacillus. *Arch. Int. Med.*, April, 1948, *81*, 501–715.

To determine the incidence of deaths from tuberculosis and of unhealed tuberculosis in persons who died from other diseases the necropsy records of Bellevue Hospital on adult patients over fifteen years of age were examined for a ten year period.

In 40 per cent of the cases involving persons over fifty years of age with unhealed pulmonary tuberculosis this disease was not mentioned in the clinical diagnosis. Of this group, 77 per cent were white males, of whom one-half had cavity formation in the lungs. A clinical recognition of the tuberculosis would not have altered the primary clinical diagnosis or the treatment prescribed in the majority of instances. However, failure to recognize the presence of pathologically active tuberculosis precluded

steps to lessen the danger of spread of the infection.

Among patients who died from tuberculosis the clinical diagnosis was incorrect six times more often for persons over fifty than for persons under thirty years. Among patients who died from other diseases, but who harbored tuberculosis which was capable of spreading, there was clinical recognition of tuberculosis in only one-fourth. Tuberculosis tends to be less explosive in type in old persons than in young adults. Older persons can have progressive disease with so few manifestations of illness that they may not seek the advice of a physician. In these circumstances they remain unrecognized spreaders of the infection.

In a high proportion of new cases of tuberculosis in adults in New York city, the disease is apparently contracted from unrecognized sources of infection. Mortality rates, encouraging as they seem do not indicate either the extent or the location of the seedbed in which the tubercle bacillus resides. The seedbed may well be greater in extent today than it was twenty years ago, for it is largely concentrated in persons, principally men, over forty-five years of age, and these are more numerous today than they were a generation ago. The seedbed must be controlled if the continuity of the disease is to be broken.—*Eugene J. McDonald, M.D.*

JONES, DEAN B. Basal pleural fluid accumulations resembling elevated diaphragms. *Radiology*, Feb., 1948, *50*, 227–233.

The occurrence of a collection of fluid between the basal surface of the lung and the superior surface of the diaphragm may yield a roentgenographic appearance simulating elevation of the diaphragm. This must be distinguished from true inflammatory or neoplastic subphrenic disease, diaphragmatic paralysis, atelectasis, herniation or eventration of the diaphragm, and intrapleural or intrapulmonary neoplastic disease.

The author presents 5 such cases and demonstrates in their differential diagnosis the utility of pneumoperitoneum, lateral decubitus roentgenograms, pleural tap, and accentuation of the stomach bubble by giving the patient a carbonated drink prior to examination.

The behavior of fluid within the pleural space is influenced by retractility or elasticity of lung tissue, capillary cohesion between the pleural surfaces, surface tension and viscosity of the fluid, and the presence of gas in the pleural space. The author believes of major importance in the basal location of fluid is the factor of retractility altered locally by fibrinoplastic adhesions of visceral to parietal layers of the pleura.—*Richard E. Lukes, M.D.*

SCHNEIERSON, SAMUEL J., and SCHNEIDER, LOUIS. Lipoid granulomatosis (xanthomatosis) with marked pulmonary fibrosis and cor pulmonale as outstanding manifestations. *Ann. Int. Med.*, April, 1949, *30*, 842–851.

Largely as a result of the contributions of Hand, Schüller, and Christian, "xanthomatosis" of the skeletal type became clearly established as a disease found principally in children. The clinical manifestations of the peculiar pathological process were described as defects in membranous bones, exophthalmos and diabetes insipidus, and were all attributable to "xanthomatous" involvement of membranous bones (chiefly skull) and of the hypothalamus.

Pulmonary infiltration has been observed by a number of authors. Most often it proves to be an incidental finding discovered in the course of chest roentgen examination, in a patient with other symptomatic manifestations of the disease. Pulmonary infiltration extensive enough to result in congestive right heart failure (as in this case) is distinctly rare. Of interest also is the fact that in a pre-employment chest roentgenogram at least two years before symptoms of the disease appeared, there was evidence of extensive pulmonary infiltration.

Confronted with such an instance, the clinician embarks on a search for etiological factors. Usually the possibility of lipoid granulomatosis is not considered. It now seems that in any instance of otherwise unexplained pulmonary fibrosis, especially in children or young adults, this entity must be kept in mind. Further interrogation of the patient and bone roentgenograms, especially of the skull, may conceivably add other cases similar to this one.

This article represents a single case of this condition. The patient is thirty-five years old, male, who had a routine chest study showing uniformly fuzzy, bilateral symmetrical infiltration throughout both lung fields. He was asymptomatic for two years when another roentgenogram of the chest showed no significant change. All laboratory studies were within normal limits. Clinical impression was that of diffuse pulmonary fibrosis, cause undetermined. He was not seen again for nearly six years by which time his condition progressively de-

teriorated. At this time roentgenographic studies of the chest revealed extensive interstitial fibrosis with enlargement of the heart to the left. Skull studies showed a large, irregular "geographic" quadralateral area of bone destruction in the right parietal region, approximately 8 by 4 cm. in size. The upper right femur also showed cystic areas of rarefaction in the shaft with thickening of the cortex. A biopsy of the lesion in the right parietal bone showed a pattern of granuloma consistent with the diagnosis of Schüller-Christian disease.

While reports of treatment by irradiation of osseous lesions in this disease have been commented on favorably by various authors, accounts of treatment of pulmonary lesions by roentgen radiation are rare. Early treatment when the pulmonary infiltration is predominantly granulomatous may be expected to be of greater effectiveness than that administered when fibrous tissue predominates, a change which is part of the evolution of this disease and one that is likely to render irradiation fruitless.—*Eugene J. McDonald, M.D.*

Lindskog, G. E., and Bloomer, W. E. Bronchogenic carcinoma. *Cancer*, July, 1948, *1*, 234–237.

The authors present a statistical comparison of two consecutive series of 100 cases of bronchogenic cancer treated by the surgical service of Yale University. These two groups are studied to determine whether or not changes in the diagnostic and therapeutic picture have occurred during the last decade.

There was a remarkable similarity in the vital statistics, symptomatology, location of primary lesion, and means of obtaining the tissue diagnosis in both series. As regards the treatment, the exploratory-thoracotomy rate has risen only slightly from 32 to 40 per cent indicating the patients are still not visiting their physicians much earlier than before. The percentage of resections has increased from 12 per cent of the total (38 per cent of all explorations) to 21 per cent (or 52 per cent of all explorations). Although this suggests progress, it more accurately reflects an increased willingness to carry out palliative resections in hopeless cases.

The summary of both series shows that in unscreened cases of bronchogenic carcinoma seen in a general hospital more than half (61 per cent) are unsuitable for exploration; that more than half of explored cases (46 per cent) will be resected, that one in five of the resections (19 per cent) will be a hospital mortality, and that less than 5 per cent of the original group will survive five years.—*J. A. Campbell, M.D.*

Agate, John N. Delayed pneumonitis in a beryllium worker. *Lancet*, Oct. 2, 1948, *2*, 530–533.

This is the history of the first known case of delayed beryllium pneumonitis in the United Kingdom although the author gives a reference listing 60 such cases elsewhere.

The patient was a physicist, aged thirty-six, engaged in lamp research. His significant exposure was between December, 1941, and December, 1942. His first symptoms occurred in November, 1945, three years after his last exposure. The condition was variously diagnosed miliary tuberculosis and sarcoidosis on the chest roentgenogram before the true cause was determined.—*J. S. Summers, M.D.*

Dotter, Charles T., and Steinberg, Israel. Angiographic study of the pulmonary artery. *J.A.M.A.*, Feb. 26, 1949, *139*, 566–572.

Changes in the pulmonary artery occur in both congenital and acquired heart disease as well as in pulmonary and other thoracic disease. Prior to the advent of angiocardiography differential diagnosis has been difficult. By this procedure the living anatomy and pathology can be demonstrated.

This paper presents an angiocardiographic study of the normal and abnormal pulmonary artery over an eleven year period.

After a detailed description of the normal pulmonary artery, including measurements, the following pathologic conditions are discussed.

(a) Congenital heart disease (the statement is made that an abnormal pulmonary artery is present in every case of congenital "cyanotic heart disease");

1. Congenital aneurysm of pulmonary artery
2. Septal defect, both auricular and ventricular
3. Patent ductus arteriosus
4. Eisenmenger's syndrome
5. Pulmonic stenosis
6. Tetrad of Fallot

All these lesions are compatible with life beyond the first few years. Angiocardiographic study is indicated in every case of the tetrad of

Fallot prior to surgery in order that surgical measures may be correctly planned.

(b) Acquired heart disease

1. Rheumatic heart disease
2. Syphilis

In all of the conditions listed under (a) and (b) distinctive and pathognomonic appearances are produced by angiocardiographic study.

(c) Pulmonary heart disease

The end result, cor pulmonale, is the same irrespective of the underlying chronic pulmonary disease among which are emphysema, fibrosis, extensive fibrotic pulmonary tuberculosis, bronchiectasis, silicosis, chronic obstruction of conus arteriosus by aortic aneurysm and primary sclerosis of pulmonary artery.

(d) Secondary abnormalities of pulmonary artery of the nature of displacement or distortion due to mediastinal and hilar masses, kyphoscoliosis, thoracoplasty, pneumothorax, and pneumonectomy.

Angiocardiography is indicated in the preoperative diagnosis of pulmonary and mediastinal tumors. Occasionally changes are demonstrated which indicate inoperability.

This is a well presented and discussed paper, adequately illustrated, which should be read in the original by those interested in this highly specialized field. As soon as a standard technique is established angiocardiography should become a routine hospital procedure. The authors make only brief mention of their technique in this discussion. They utilize standard equipment and rely on two chest films produced at predetermined times after the intravenous injection, with an ordinary stereoscopic cassette changer. Many different techniques have been used by others such as photofluorography, cinefluorography and rapid serial study with full sized films and cassettes.—*Frank Huber, M.D.*

Nemec, Stanley S. Differential diagnosis of retrocardiac shadows. *Radiology*, Feb., 1948, *50*, 174–183.

Variations in density within the cardiac shadow may present characteristic features of diagnostic importance and aid in early detection and proper differentiation and evaluation of asymptomatic retrocardiac lesions. Moderate overpenetration of chest roentgenograms aids in the differential diagnosis of lesions not obvious on routine exposures.

Herniation of the stomach through the diaphragm may produce a duplication of density within the cardiac silhouette. Barium meal confirms the diagnosis. The author outlines a double contrast method of better visualizing the lower esophagus and stomach in obese patients whose stomach is retrocardiac and difficult to demonstrate. The fasting stomach is first distended with carbon dioxide from cold soda water, and barium then administered, followed by proper fluoroscopic and film examination. The cardio-esophageal junction is often well demonstrated by displacing fundus gas into the lower esophagus by a horizontal or Trendelenburg oblique or left lateral decubitus position, and trapping this gas with an additional barium swallow.

Right-sided widening of the mediastinum, or reduplication of the right cardiac contour, and obliteration of the right cardiophrenic angle, are characteristic of megaesophagus; whereas left-sided widening is produced predominantly by aortic aneurysm, tortuous aorta, and retrocardiac stomach. Large esophageal diverticula, and mediastinal enteric cysts may produce retrocardiac shadows.

Among diseases of the lung producing retrocardiac shadows are atelectasis, bronchogenic cysts, tuberculoma, echinococcus cyst, pneumonia, bronchiectasis, lung abscess, and mediastinal pleural effusion. Vertebral column disease includes tuberculosis of the spine with paravertebral abscess, scoliosis, sympathicoblastoma, fibrosarcoma, and tumors of neurogenic origin. Cardiovascular conditions discussed were aneurysm of the descending aorta, congenital and senescent elongation and tortuosity of the aorta, and left atrial enlargement. —*Richard E. Lukes, M.D.*

Gross, Robert E., and Hurwitt, Elliott. Cervicomediastinal and mediastinal cystic hygromas. *Surg., Gynec. & Obst.*, Nov., 1948, *87*, 599–610.

Cystic hygroma of the neck is an uncommon lesion but a well recognized entity. In view of the marked tendency of these cystic masses to involve adjacent structures by direct extension one might anticipate a relatively high incidence of cervicomediastinal lesions. However, a review of the literature reveals only 19 cases of cervicomediastinal hygromas while intrathoracic hygromas without involvement of the neck are even more rare. Cystic hygromas are congenital malformations. Characteristically, they are thin-walled cystic structures lined

by endothelium. The authors report 3 cases.

In the tumors with mediastinal involvement the roentgenogram may demonstrate a slightly lobulated, smoothly outlined mass. It is usually not possible by this means alone to distinguish hygromas from other benign tumors or cysts of the mediastinum (such as duplications of the esophagus, bronchial cysts, dermoids, and teratomas, pericardial celomic cysts or tumors of the thymus gland).

The authors recommend treatment of cervicomediastinal hygromas by (1) separate operations in the neck and thorax, and (2) by excision of the cervical portion and simultaneous sclerosis of the intrathoracic part. However, they call attention to the favorable results reported by Singleton and Goetsch wherein excision of the cervical portion was followed by roentgen therapy to the mediastinum and also the procedure described by Portmann wherein radon seeds were applied to the mediastinal remnants.—*Mary Frances Vastine, M.D.*

Sengpiel, Gene W., Ruzicka, Francis F., and Lodmell, Elmer A. Lateral intrathoracic meningocele. *Radiology*, April, 1948, *50*, 515–520.

The authors point out rarity in that only 3 cases were reported from 1920 to 1946 in which meningocele originated from the lateral aspect of the spine, and report such a condition occurring in a twenty-one year old soldier.

In the reported case the skeletal defect was minimal. The skeletal defect appears to play the role of a necessary concomitant in the development of a meningocele. Embryology of the meninges also plays important part in development of meningocele and same is discussed.

Importance of differential diagnosis of solitary, rounded, well circumscribed lesions appearing in the chest. Importance of a diagnostic pneumothorax is demonstrated. In retrospect usual myelographic procedures would have established the diagnosis and avoided necessity of an operation.—*Robert D. Moreton, M.D.*

ABDOMEN

Whitehouse, Francis R., and Kernohan, James W. Myenteric plexus in congenital megacolon. *Arch. Int. Med.*, July, 1948, *82*, 75–111.

The myenteric plexus has been studied in 11 cases of congenital megacolon, a series of cases used as controls and 5 cases of secondary megacolon.

The sympathetic nervous system is generally conceded to have an inhibitory action on the colon and thus causes retention of the fecal contents and retards the emptying of the colon. The parasympathetic nervous system has an augmentative action on the colon that causes evacuation of the fecal contents and accelerates the emptying of the colon. The internal anal sphincter will relax after parasympathetic stimuli and undergo an increase in tone after sympathetic stimuli. With such a physiologic basis, it has been postulated that the cause of the fecal retention in cases of megacolon is an overactivity of the sympathetic nervous system in the presence of a normally active parasympathetic nervous system.

The myenteric plexus was found to be absent in the most distal part of the colon in all cases of congenital megacolon. In 80 per cent of the cases it was absent also in the "transitional region." In 60 per cent of the cases it was in addition absent in the lower part of the sigmoid. In 20 per cent of the cases the absence of the myenteric plexus extended from the rectum into the upper part of the sigmoid and the descending colon. In all cases of congenital megacolon there were nerves present in the location of the myenteric plexus which were not seen in the control cases.—*Eugene J. McDonald, M.D.*

Culver, Gordon J., and Kline, J. Richard. Acute gaseous cholecystitis. *Radiology*, April, 1948, *50*, 536–538.

Three good figures demonstrating three of McCorkle and Fong's four criteria for roentgen diagnosis of gaseous cholecystitis, namely, nonvisualization of the duct system, a gallbladder of normal size, and gas in the gallbladder wall.

This patient had severe non-radiating right upper quadrant pain, jaundice, fever and laboratory evidence of gallbladder obstruction. Responded favorably to penicillin and sufadiazine. The authors advocate conservative treatment and believe even interval cholecystectomy is contraindicated.—*E. E. Seedorf, M.D.*

Janus, William L. Regional jejunitis. *Radiology*, April, 1948, *50*, 532–535.

The author reveals that only 18 examples of regional enteritis of the upper small bowel had appeared in the literature until 1939. He asks the question whether the condition is rare or merely unrecognized. He presents 3 cases of regional jejunitis of which 2 were diagnosed

preoperatively by roentgen examination.

In early stages irritability and hypermotility of the involved portions of the bowel are characteristically present. Suspected areas must be observed fluoroscopically and studied with spot films in addition to routine progress films. A chest film is essential to complete the diagnosis in all suspected cases of regional jejunitis since it may be simulated by tuberculosis. Sprue and vitamin deficiency states must also be considered in the presence of a disturbed small bowel pattern.

The author emphasizes fact that regional enteritis is not necessarily limited to the ileum, nor must it involve the ileum and proceed toward the stomach.—*Robert D. Moreton, M.D.*

Feldman, Maurice. Statistical study of life cycle of 1,154 cases of duodenal ulcer. *J.A.M.A.*, March 13, 1948, *136*, 736–738.

The causes of peptic ulceration remain a debatable question. The role of acid still seems predominant in the etiology of this condition. The importance of psychogenic influences is fairly well established, but the exact mechanism which it plays in the etiology of peptic ulcer is not definitely known. Recent studies on vagotomy have stimulated considerable interest in this condition and emphasized the neurogenic influence in peptic ulcer.

This study involving 7,300 gastrointestinal examinations was made on an unselected group of ambulatory private patients of both sexes and all ages presenting digestive disturbances. These cases were divided into two five year periods: (1) prewar, 1937 to 1941 (3,500 cases), and (2) during and immediately after World war II, 1942 to 1946 (3,800 cases). An appraisal of the data collected over a ten year period, with two separate psychological periods, should give some statistical evidence regarding the cycle of peptic ulcer and materially add to our knowledge. During this ten year period, 1,154 cases of duodenal ulcer were found, an incidence of 15.87 per cent. In the five prewar years there were 480 cases, or 13.7 per cent. In the second five years, during and immediately following the war, there were 674 cases of duodenal ulceration, an incidence of 17.7 per cent.

The following information is revealed by this study: 1. A rising trend in the incidence of duodenal ulcer during and immediately after World War II amounted to about 4 per cent. 2. The comparison of the age and sex incidence in duodenal ulcers between the prewar and the war period showed no significant statistical change. 3. There was a slight increase in the incidence of cases of less than one year duration in the second five year period. 4. According to the objective roentgen findings for the determination of the activity of the ulcer, there seemed to be an increase in the second five year study. 5. There was slight increase in the number of niches observed roentgenologically in the wartime cases. A slight increase was noted in the number of niches occurring in females in the war group. 6. There was a higher incidence of first recurrences and an increase in the incidence of "acute" ulcers during the war period. 7. In many instances the severity of symptoms seemed to be more pronounced during the war period, because of the increased number of acute ulcers. 8. The complications, obstructions and hemorrhages were not statistically different in the two groups of cases. There was a greater incidence of hematemesis in females during the war period.—*C. W. Williams, M.D.*

Rafsky, Henry A., Weingarten, Michael, and Krieger, Charles I. Onset of peptic ulcer in the aged. *J.A.M.A.*, March 13, 1948, *136*, 739–741.

From 1,800 consecutive admissions for gastric ulcer, the authors have selected 81 cases (4.5 per cent), which represent those with initial ulcer symptoms occurring after the fifth decade of life. Of these, 60 patients were male, and 21 were female. Forty-seven of the group presented complications on admission, the main symptom of which was hemorrhage, with 3 being associated with perforation. Of the uncomplicated cases, the outstanding symptom was pain. Attention is called to the difficulty in differentiating benign and malignant lesions in this age group with symptoms of short duration. Gastroscopic examination is advised whenever feasible. Treatment employed was mainly medical, with only 19 of the group being treated surgically. Of these, 11 had perforation, 6 of which died. The mortality rate in this group of patients was 16 per cent (11 patients). The authors feel that gastric ulcer in the aged is not infrequent, and that complicating diseases play a very important role in the prognosis.—*C. Sory, M.D.*

Walters, Waltman, Neibling, Harold A., Bradley, William F., Small, John T., and Wilson, James W. Results of vagus nerve

resections in treatment of peptic ulcer. *J.A.M.A.*, March 13, 1948, *136*, 742–747.

The cases in this report are divided into two series all of which were performed at the Mayo Clinic:

Series I (Performed by the Authors).

Vagotomy Alone: Vagotomy alone was done in 10 patients with duodenal ulcer, in 2 with gastrojejunal ulcer, and in 5 with gastric ulcer. All obtained relief of the pain caused by ulcer. Three of the 10 patients with duodenal ulcer, and 1 of the 5 with gastric ulcer had disturbances of motility. Four of the patients with duodenal ulcer were reported at dismissal as still having visible ulcers on roentgenologic examination. Vagotomy with a simultaneous gastric operation was done in 24 patients with duodenal ulcer, in 5 with gastrojejunal, and in 4 with gastric ulcer. In 23 of the 24 patients with duodenal ulcer, all 5 with gastrojejunal ulcer, and in 2 of the 4 patients with gastric ulcer, relief of distress from the ulcer was obtained. Ulceration recurred in 1 patient with gastric ulcer in whom subtotal gastrectomy was necessary six months later, and in another the gastric ulcer failed to heal. Both had pronounced reductions of gastric acidity and repeatedly negative reactions to the insulin test after vagotomy. Six of the 24 patients with duodenal ulcer who had associated gastroenterostomy, 2 of 5 with gastrojejunal ulcer, and 3 of 4 patients with gastric ulcer all had moderate to marked disturbances of motility.

Series II (Colleagues' Cases).

Duodenal Ulcer: There were 8 cases having vagotomy only, 5 of these were done transthoracically, and 3 abdominally. Of the 5 done by the transthoracic approach, 4 had good results, although 2 had some retention of food and fluid postoperatively. The fifth patient complained of gas, and occasional episodes of diarrhea eight months after operation. Of the 3 who underwent vagotomy abdominally, 1 was well at dismissal. Another had intermittent vomiting and diarrhea three months after surgery, and the third died postoperatively apparently from pulmonary embolism.

Vagotomy and gastroenterostomy were done in 25 patients with duodenal ulcer. Twenty-four had relief of the symptoms of ulcer. Eighteen of these 24 had no disturbances of motility. Six patients had disturbances of motility characterized by epigastric fullness, nausea and diarrhea. The 25th patient died on the fourteenth postoperative day from perforated duodenal ulcer and subdiaphragmatic abscess.

Vagotomy and Pyloroplasty: There were 4 patients in this group. All obtained relief of distress from ulcer. Two had disturbances of motility of consequence six and ten months postoperatively.

Gastrojejunal Ulcer: There were 30 cases in this group but in 3, the operation was performed too recently to permit evaluation. Of the 27 cases, 15 were done transthoracically and 12 abdominally. Of the 15 done transthoracically, 9 had good results from vagotomy with absence of all symptoms of ulcer. Three patients had unsatisfactory results with recurrence of the symptoms of ulcer. Of 7 done abdominally, the existing anastomosis was not disturbed. Of these, 3 had obtained good results clinically when dismissed. Three still complained of minor degrees of fullness and diarrhea several months after operation. The remaining patient noticed gas and dizziness when hungry which was relieved by frequent small meals. The remaining 5 had good results.

Gastric Ulcer: There was 1 patient in this group. Along with vagotomy, the ulcer was excised and pyloroplasty done simultaneously. Early relief of symptoms was obtained with a reduction in acidity.—*C. W. Williams, M.D.*

MARSHALL, SAMUEL F., and WELCH, MARK L. Results of surgical treatment for gastric ulcer. *J.A.M.A.*, March 13, 1948, *136*, 748–751.

Gastric ulcer presents a serious problem in its treatment because of possible malignant changes arising in a benign ulcer and especially because of the considerable percentage of diagnostic errors made in distinguishing between benign and malignant ulceration of the stomach.

At the Lahey Clinic, from 1936 to 1945, inclusive, in those cases in which a preoperative diagnosis of benign gastric ulcer had been made, partial resection was carried out on 131 patients. The histopathologic examination of the removed lesions disclosed carcinoma in 26 of the 131 cases, a diagnostic error of 19.8 per cent in the cases in which operation was done. During this same ten year period approximately 8,000 patients with peptic ulcer were treated. Of this number, 800 had gastric ulcers. In the entire group of 800 cases treated medically and surgically, there were 26 proved malig-

nant gastric ulcerations, an incidence of proved malignancy of 3.3 per cent for the entire group. This is assuming that none of the patients treated medically proved later to have cancer, a most unlikely possibility. All of this group of 131 cases were, however, ultimately submitted to operation by the gastroenterologist because of failure to heal, and because, therefore, gastric cancer could not definitely be ruled out. Seventeen of this group of 26 proved malignancies had been treated one month or longer before decision to operate was made. This trial period of observation over a month is considered to be too long. Decision regarding necessity of surgical intervention should be made much earlier.

The results following adequate partial gastrectomy for benign gastric ulcer (105 cases in this series), are excellent. Eighty-six per cent of the patients had absence of free hydrochloric acid after operation. There were no postoperative recurrent ulcers and no obstruction occurred in any case at the gastrojejunal stoma. No attempt should be made to treat any patient with gastric ulceration by resection of the vagus nerves because of the difficulty in determining whether an ulcer is benign or malignant. This can be determined definitely only by pathologic section.

Most patients within a few weeks or months after the operation can eat an ordinary, full diet with few restrictions and with no gastric distress. All are relieved of ulcer distress and pain, and all are able to work usually within three months after operation.

There were three deaths following resection, an operative mortality of 2.8 per cent, in the group of 105 cases with benign gastric ulcer. Two deaths were due to pulmonary embolism, and one followed coronary occlusion.—*C. W. Williams, M.D.*

Thompson, Harold L. Complications of peptic ulcer. *J.A.M.A.*, March 13, 1948, *136*, 752–761.

The author lists as primary complications of peptic ulcer: (1) perforation, (2) hemorrhage, (3) obstruction, (4) intractability, and (5) malignant degeneration. This paper is concerned with the first three.

Pyloric obstruction may be associated with the acute phase of a duodenal ulcer, resulting from edema and spasm, and is considered a medical problem, responding to routine medical management. If the obstruction results from cicatricial formation, surgery is employed. Differentiation is mainly clinical. Surgical treatment advised is subtotal gastrectomy. Vagotomy with gastrojejunostomy seems to give good results, but at the present time may well be limited to treatment associated with acute ulcers.

Hemorrhage from peptic ulcers is a common complication, and the choice of medical or surgical treatment should be made through consultation with a competent internist and surgeon. Emergency surgery should be considered only in cases of acute hemorrhage and of a relatively severe nature. The age of the patient with a bleeding peptic ulcer is considered an important deciding factor, and the author suggests a trial of medical treatment for those under forty-five years of age, and favors the surgical approach for those over forty-five years. Those in the upper age group are more likely to have other complicating diseases which do not lend themselves favorably to systemic effects resulting from hemorrhage. If surgery is anticipated, it is advised within the first forty-eight hours.

Perforation of a peptic ulcer is considered as an acute surgical emergency, with the mortality rate increasing with increase in time between perforation and surgery. Increase in the amount of food in the stomach at the time of perforation also increases the mortality rate. The author favors closure of the perforation, cleaning the peritoneal cavity, and closure of the abdominal wound without drains. He points out that in his experience the mortality rate is increased in direct proportion to the number of drains used. He feels that gastric resection should be reserved as a second stage procedure, if needed, and when the patient is in better condition.—*C. Sory, M.D.*

St. John, Fordyce B., Harvey, Harold D., Ferrer, Jose M., and Sengstaken, R. W. Results following subtotal gastrectomy for duodenal and gastric ulcer. *Ann. Surg.*, July, 1948, *128*, 3–14.

In an analytical review of 394 partial gastric resections for peptic ulcers the authors make the following points: (1) There has been a high percentage of excellent results with the patients returning to a normal life. Recurrences have been infrequent. (2) For individuals under forty-five years the mortality has been about 1 per cent. (3) In the older age group, if there is any suspicion of carcinoma at all, resection

should be carried out. In cases of bleeding duodenal ulcer in the older patients the lower mortality of vagus resection is appealing. However, follow-ups on these cases have not been long enough for adequate evaluation. (4) Psychic factors have not appeared to seriously interfere with the benefits of surgery. Many patients have lost their psychoneurotic tendencies when free of pain or bleeding. (5) There has been a steady improvement in the postoperative mortality at the Presbyterian Hospital, New York City, in the past thirty years dropping from 20 per cent in 1916–1925 to 2.5 per cent for the years 1946–1947.—*T. D. Allison, M.D., and C. L. Hinkel, M.D.*

Moore, George E., State, David, Hebbel, Robert, and Treloar, Alan E. Carcinoma of the stomach; the validity of basing prognosis upon Borrmann typing or the presence of metastases. *Surg., Gynec. & Obst.*, Nov., 1948, *87*, 513–518.

An analysis, according to Borrmann's classification, of the relative influences of regional metastases and the gross anatomic types of gastric carcinoma on postoperative survival is presented in this paper.

Schindler has recently offered evidence that the prognosis and perhaps the advisability of any therapeutic procedure for carcinoma of the stomach may be based upon the Borrmann type of the tumor. Borrmann separated the gastric carcinomas into 4 gross types: I, sharply demarcated polypoid carcinomas; II, sharply demarcated ulcerated carcinomas; III, partly infiltrating carcinomas; IV, diffusely infiltrating carcinomas. Combining Types I and II as "limited" and Types III and IV as "infiltrative" groups, Schindler noted that there was a much higher resectability rate and lower mortality rate for the former group. On the basis of his data, Schindler questioned the advisability of resection of infiltrative tumors.

The authors point out that survival is dependent on complete removal of the tumor and the likelihood of such removal should, in the case of the stomach, just as for other organs, be to a degree reflected in the presence or absence of regional metastases. Consequently, they have compared the effects of the gross anatomic types of carcinoma and regional metastases upon the survival rates.

Conclusions.

1. The presence or absence of demonstrable metastases in regional nodes among the patients subjected to gastric resection for carcinoma has a greater prognostic value than does the Borrmann type of the tumor.

2. The Borrmann type affects the prognosis of groups of cases to the extent that the limited tumors provide a proportionately large number of patients free of metastases while the infiltrating tumors which are more numerous are more likely to have metastasized.

3. The presence or absence of metastases cannot be accurately determined preoperatively and no patient should be denied operation because of the type of tumor he may be diagnosed as having.—*Mary Frances Vastine, M.D.*

Beynon, A. E. Primary carcinoma of the liver in a boy aged 15. *Lancet*, Oct. 2, 1948, *2*, 528–530.

This is an unusual case first detected because of metastatic tumor seen in the lung fields on a mass survey. The case is of interest because of the lack of symptoms and the maintenance of body weight for a year after the tumor was first discovered. There was but slight increase in the size of the lung lesions.

The diagnosis was definitely made by aspiration biopsy. The boy died after sixteen months of observation. There was never any evidence of icterus. Necropsy was refused.—*J. S. Summers, M.D.*

Nickell, David F., and Dockerty, Malcolm B. The five year survival rate in cases of completely obstructing annular carcinoma of the descending colon and sigmoid; pathologic study. *Surg., Gynec. & Obst.*, Nov., 1948, *87*, 519–524.

The five year survival rate in 37 cases of completely obstructing annular carcinoma of the descending colon and sigmoid in which resection was performed is compared with the five year survival rate in cases in which nonobstructing lesions had been treated surgically. These rates were 25.7 per cent and 51.7 per cent respectively.

1. The five factors which may contribute to the lowered five year survival rate are: (1) damage to the intestinal wall with increased permeability and increased lymphatic absorption; (2) hypertrophy of the musculature which produces a massaging and traumatizing action to the carcinoma at the site of obstruction; (3) inflammatory swelling and edema which cause breakdown of any mechanical protective

factor, and of any protective forces elaborated the host; (4) the invasive property of the cancer cell, whether it be motility or some lytic action, which is facilitated by the effect of the factors just mentioned, and (5) the ileocecal valve which may produce a closed-loop type of obstruction.

2. The size of the lesion is of no significance in prognosticating the presence of metastasis and the volume of malignant tissue is actually less in the cases of obstruction than in the cases in which no obstruction was present.

3. No correlation was found between the grade of the lesion and the incidence and degree of obstruction or between the duration of obstruction in days and the survival rate.

The authors point out that this is a small series of cases on which to base any definitive conclusions but with the strict criteria for obstruction used, namely, complete obstruction requiring a colostomy or cecostomy for decompression before removal of the lesion, the series would of necessity have to be small. Notwithstanding, they believe that such a difference in the five year survival rate in cases of obstructing and nonobstructing carcinoma of the descending colon and sigmoid, 26 per cent in this series, is significant. This difference is not due to a difference in the grade of malignancy of obstructing carcinomas but rather apparently to the condition of the intestinal wall attending the complication of obstruction. —*Mary Frances Vastine, M.D.*

BLACK, WILLIAM A., and WAUGH, JOHN M. The intramural extension of carcinoma of the descending colon, sigmoid and rectosigmoid; pathologic study. *Surg., Gynec. & Obst.*, Oct., 1948, *87*, 457–464.

The present-day approach to the cure of carcinoma of the colon is surgical extirpation of the primary lesion and all its extensions with restoration of continuity of the bowel and preservation of sphincteric control whenever feasible.

The importance of carcinoma of the colon is attested by its incidence and cause of death in the general population. The mortality statistics of the Bureau of Census for 1936 give the total death rate from all causes as 1,151.8 per 100,000 population. Of these deaths, cancer and other malignant tumors cause 111 per 100,000. Of the deaths due to cancer, cancer of the intestines except the duodenum, rectum, and anus accounted for 12 per 100,000 population. Since less than 3 per cent of carcinomas of the intestines originate in the small bowel, the figure for cancer of the intestines represents essentially that for carcinoma of the colon, exclusive of the rectum and anus.

Conclusions.

1. In carcinoma of the colon, the raised edge of the lesion may not mark the limit of intramural extension.

2. There is no appreciable difference in intramural spread of carcinoma above or below the lesion.

3. Intramural spread of carcinoma is greater in the descending colon than in other sites in the left portion of the colon.

4. The plane of greatest extension in the bowel wall is the submucosa.

5. The grade of malignancy (Broders' method) of the lesion has no relation to the degree of intramural spread.

6. Spread of carcinoma through the bowel wall appears to follow the course of the intramural lymphatics.

7. Spread of carcinoma to the extramural lymphatics occurs before the entire bowel wall is penetrated.

8. For resections of the left part of the colon for carcinoma, only 2 centimeters of normal bowel need be allowed above and below the lesion in order to remove the whole of the primary lesion.—*Mary Frances Vastine, M.D.*

GENITOURINARY SYSTEM

HENLINE, ROY B., and HAWES, CECIL J. Ureteropelvic obstructions; symptoms and treatment. *J.A.M.A.*, June 26, 1948, *137*, 777–784.

Gastrointestinal symptoms may be the first or sole manifestation of an obstructive renal syndrome. Due to the common nerve supply of the kidney and gastrointestinal tract the symptoms of indigestion, nausea and flatulence may result from kidney disease even though renal symptoms are absent and urinary studies are normal. In this report of 52 patients with ureteropelvic obstruction, the presenting complaints in 20 were of a gastrointestinal nature. Renal pain, such as backache and colic, was present in 46 cases and urinary symptoms in 22. It is interesting to note that in 19 patients the voided urine was normal.

The most common cause of ureteropelvic obstruction is intrinsic stenosis due to stricture, to hypertrophy of the circular muscle and valves

or to the presence of fibrous tissue. In 62 kidneys which were operated upon, 71 per cent showed obstruction caused entirely by intrinsic disease, while extrinsic factors alone, such as aberrant vessels, bands and adhesions were present in 3.2 per cent. A combination of intrinsic and extrinsic factors was observed in 25.8 per cent. The importance of these findings cannot be overestimated inasmuch as the correction of an obvious external cause such as separation of vessels or bands results in failure of the operation if intrinsic pathology is overlooked.

Although excretory urography frequently demonstrates ureteropelvic obstruction it is the practice of the authors to rely only on the ten minute delayed roentgenogram following retrograde pyelography to make the diagnosis. Dilatation of the internal structure of the kidney and retention of dye are observed.

Formerly, partial ureteropelvic obstruction with accompanying hydronephrosis was treated by nephrectomy. With increasing knowledge of the etiologic factors, improvement in surgical technique and the use of antiseptics and antibiotics, plastic operations now result in the preservation of renal tissue.

In the treatment of ureteropelvic obstruction the authors prefer the Foley Y plastic operation, employing a nephrostomy tube for drainage and a soft rubber splinting catheter passed halfway down the ureter. The catheters are left in place for six weeks. By this method of treatment there were no operative deaths and relief of symptoms was obtained in 87.2 per cent of the patients.—*Gerald Lavner, M.D.*

IRVIN, G. E., and KRAUS, JOHN E. Congenital megaloureter and hydroureter; pathogenesis and classification. *Arch. Path.*, June, 1948, *45*, 752–765.

After briefly reviewing the history of the subject and citing the various etiological theories previously advanced, the authors conclude that there are three basic types. True megaloureter represents hyperplasia of all elements of the ureteral wall without evidence of obstruction or neurologic defect, resulting in a thickened tortuous ureter with a nearly normal lumen. This may be associated, as in their case, with megalobladder and hydronephrosis. A second type is a simple dilatation from organic obstruction or neurologic defect. Third, they distinguish an intermediate type: secondary dilatation of a true megaloureter, of which they present 2 cases, both with megalobladder and hydronephrosis.

The authors emphasize the view that true megaloureter is probably a congenital structural anomaly rather than the result of disturbed ureteral physiology.—*D. D. Beiler, M.D. and C. L. Hinkel, M.D.*

HERBST, WILLIAM P. The posteriorly located renal pelvis associated with ptosis. *J.A.M.A.*, June 26, 1948, *137*, 775–777.

In the author's experience, patients with ptosis of the kidney and with symptoms severe enough to warrant surgical treatment, all showed the renal pelvis coming from a scooped-out-like depression on the posterior aspect of the kidney. The diagnosis of posteriorly located renal pelvis was made in 25 patients, of whom 19 were operated on. This abnormality is the result of fusion between the ureteral bud and metanephros posteriorly, rather than of an anomaly of rotation of the kidney. The roentgenographic signs are inconstant but the diagnosis is suggested in the anteroposterior projection by (1) the appearance of a gun barrel effect in the ureteropelvic region, (2) the location of the ureter lateral to the medial pelvic border, and (3) in the lateral and oblique projections by visualization of the ureter arising posteriorly. The treatment of the posteriorly placed urinary pelvis associated with ptosis is nephropexy, primary nephrectomy, or resection of the lower pole of the kidney with removal of the overhanging mass of renal tissue.—*Gerald Lavner, M.D.*

ABOWITZ, JACOB. Obstructive hydronephrosis produced by aberrant blood vessels and diagnosed by intravenous urography. *Radiology*, Jan., 1947, *48*, 33–36.

This article is a brief general discussion of the subject of ureteral obstruction by anomalous blood vessels which supply the kidney. The author properly indicates the important place of excretory pyelography in the diagnosis of this condition.

The roentgen findings may be divided into three general classifications: The first represents the caliceal and pelvic dilatation due to the obstructive uropathy and include the evidence, as indicated in the erect film, that the upper urinary tract does not empty its contents properly. The second group of roentgenographic findings are those due to the defect produced in the ureter by the aberrant vessel or by the fibrot-

ic bands which represent atresia of an aberrant vessel. These findings on the urogram are described by the author as a sharp line of decreased density, indicating the site of the constriction produced by the aberrant vessels.

A third group of roentgen findings may be considered as the advanced hydronephroses usually associated with an extrarenal type of pelvis in which the cause of the obstruction is obliterated by the marked dilatation of the renal pelvis and by the consequent hydronephrosis and atrophy of the kidney.—*George W. Chamberlin, M.D.*

Rudhe, Ulf. A typical roentgen picture of very large ureteroceles. *Acta radiol.*, 1948, *29*, 396–402.

During the years of 1946 and 1947 three cases of very large ureteroceles have been observed at the roentgen diagnostic department of the Caroline Hospital. The diagnosis in each case was confirmed at cystoscopy or operation. As the roentgen picture in such cases appears to be little known, the cases are described in detail.

These ureteroceles appear as large, rounded, distinctly outlined soft tissue formations which cause corresponding defects in the bladder contrast. In 2 of the cases the picture resembled that of prostatic hypertrophy. Two of the cases showed the distal ends of the ureters to be angulated in the shape of a hook with the ureteral orifices elevated. This appearance has also been described as typical of prostatic hypertrophy. There were no signs of trabeculation in the bladder wall. (In cases of prostatic hypertrophy of this size the wall is usually trabeculated because of the chronic obstruction to emptying.) In ureterocele, this same trabeculation may occur. The ureterocele, if large, may reach the internal vesicle orifice and act as a valve over the bladder outlet.

Anomalies such as double renal pelves and ureters are rather common in cases of ureterocele. In the 3 cases reported completely duplicated renal pelves and ureters were found on the same side as the ureteroceles. The ureteroceles involved the ureters belonging to the upper pelves. Stenosis at the intravesical ureteral orifice brought about dilatation of the ureter and upper renal pelvis resulting in loss of function in the kidney parenchyma.

In conclusion, the author emphasizes that the ureteroceles in cases with coexisting complete reduplication of renal pelvis and ureters, as a rule, seem to affect the ureter from the upper pelvis.—*Mary Frances Vastine, M.D.*

Perry, S. Paul, and Haden, W. Dan. Accidental filling of urinary tract during barium enema. *Radiology*, April, 1948, *50*, 539–540.

A case report in which the complete urinary tract became visualized with barium during routine fluoroscopic examination of the colon.

Several years previously the patient had a perforated sigmoid diverticulum; then a rectovaginal fistula developed and recently the patient had been passing feces and gas when urinating.

Patient experienced no ill results. A subsequent intravenous pyelogram was normal.—*E. E. Seedorf, M.D.*

NERVOUS SYSTEM

Brierley, J. B., and Field, E. J. The connexions of the spinal sub-arachnoid space with the lymphatic system. *J. Anat.*, July, 1948, *82*, 153–166.

The authors, after reviewing the literature, present their techniques for studying the subarachnoid connections in adult rabbits, using india ink as an indicator. They present their observations and discussions in a comprehensive manner. They have demonstrated that under conditions as nearly physiological as possible, ink particles not over 1.5 microns can pass readily from the subarachnoid space into cervical and prevertebral lymph nodes. These latter may be regarded as the regional lymph nodes of the subarachnoid space. Their studies show that ink introduced into the subarachnoid space appears in the nasal mucosa and cervical lymph nodes within four hours. Ink introduced into the cranial subarachnoid space can be found as low as the mid-thoracic level at the end of one hour and throughout the spinal subarachnoid space in six hours. Concentration of ink is marked around the lumbosacral nerve roots and in the terminal part of the spinal subarachnoid sac. Lymph nodes around the aortic bifurcation and in the hollow of the sacrum show ink filling in six hours. Repeated ink injection produces filling of the lymph nodes arranged along the front of the thoracic and lumbar vertebrae in a more or less "segmental" manner. The pathway of the outflow is discussed.—*H. G. Reineke, M.D.*

Naffziger, H. C., and Boldrey, E. B. Cancer of the nervous system; brain spinal cord and

peripheral nerves. *J.A.M.A.*, Jan. 10, 1948, *136*, 96–103.

Tumors of the nervous system constitute 2 per cent of all cancers. Gliomas constitute 50 per cent of all intracranial tumors and must be regarded as malignant. Astrocytomas are the most common glioma and these types are recognized: fibrous, which carries the best prognosis; protoplasmic, a diffuse type which is fairly responsive to roentgen therapy; and gemistocytic, which is usually rapidly fatal. Second most common of the gliomas is glioblastoma multiforme, the sudden onset of which may lead to an erroneous diagnosis of a vascular accident. Combined surgery and roentgen therapy may give survival rates as long as five years after the first diagnosis. Medulloblastoma, the commonest glioma of childhood should never be regarded as being completely removed by surgery, and since the cells are radiosensitive, judicious use of roentgen therapy following surgery is the treatment of choice. Survival up to five years is not uncommon. Ependymoma constitutes about 10 per cent of all gliomas and is fairly resistant to roentgen therapy. Polar spongioblastoma constitutes about 5 per cent of all gliomas, is most often seen in the region of the pons, optic chiasm and corpus callosum, which fact usually makes this tumor not amenable to surgery. It is moderately susceptible to roentgen therapy and patients have been carried along successfully for several years by its judicious use. Oligodendroglioma constitutes about 5 per cent of all gliomas, is relatively indolent and but moderately responsive to roentgen therapy.

Meningiomas constitute about 16 per cent of brain tumors. They usually arise from the arachnoid, and are frequently associated with increase in vascular supply which may be manifest in plain roentgen films of the skull. Usually, they are resistant to roentgen therapy.

Cerebellopontine angle tumor (acoustic neuroma) constitutes about 10 per cent of intracranial neoplasms. They may be bilateral and are sometimes associated with von Recklinghausen's disease.

Pituitary tumors constitute about 5 per cent of all intracranial tumors. Chromophobe, eosinophilic and basophilic adenomas are discussed briefly. The authors favor, in general, attempted surgical removal prior to roentgen therapy, if visual acuity is threatened.

Tumors of the hypophysial duct, epidermoid and dermoid cysts, angiomas and hemangioblastomas are discussed briefly. The latter is susceptible to roentgen therapy to a moderate degree.

Intracranial granulomas are also mentioned since they may produce symptoms indistinguishable from neoplastic growths.

A brief discussion of the signs and symptoms of neoplasm of the spinal cord is presented with the statement that 50 per cent are completely removable with anticipation of a good result if surgery is done prior to irreparable damage to the cord. The tumors which are discussed very briefly are extramedullary tumors, meningioma, perineural fibroblastoma, gliomas (include all the types mentioned in the discussion of intracranial gliomas), angioma, epidermoid cysts, intrinsic tumors of the vertebrae exerting pressure on the cord by direct extension and intervertebral disc.

Tumors of the peripheral nerves are almost always tumors of the nerve sheath, such as perineural fibroblastoma (which usually can be enucleated), neurofibromas and neurofibrosarcomas. Tumors of the neuronal element are exceedingly rare. The ganglioneuromas, seen usually in infants and childhood, are encapsulated and if completely removed, do not tend to recur. But the rare neuroepithelioma is highly malignant and requires radical treatment.—*M. M. Manalan, M.D.*

SKELETAL SYSTEM

Meschen, Isadore, and McGaw, W. H. Newer methods of pneumoarthrography of the knee with an evaluation of the procedure in 315 cases. *Radiology*, Dec., 1947, *49*, 675–711.

The authors have presented an analysis of their experience with 782 pneumoarthrograms of the knee performed between April, 1942 and April, 1945. The examination is believed to be without risk and is now done routinely in all cases of suspected internal derangement.

Technique.

Oxygen Insufflation: 80–120 cc. of oxygen is injected through a needle inserted into the joint space along the lateral aspect of the knee just below the junction of the quadriceps tendon with the superior articular margin of the patella. This is done under strict asepsis. After the injection the patient is turned to the prone position and the knee flexed and massaged gently

for a few minutes to circulate the oxygen evenly through the joint.

Roentgenological Examination: It is important that the tibial plateau is perpendicular to the film and that the central ray goes through the joint space. A mechanical device is described which facilitates widening the lateral and medial aspects of the joint. Five views are usually obtained: (1) anteroposterior with spreading of the medial side of the joint; (2) anteroposterior with spreading of the lateral side of the joint; (3) posteroanterior with spreading of the medial side of the joint; (4) posteroanterior with spreading of the lateral side of the joint; (5) a straight lateral with the knee partially flexed. If these views do not give sufficient information, excellent visualization can be obtained by turning the knee on one side or the other and allowing it to rest on a block which acts as a fulcrum when it is spread. The roentgenograms are then taken by a horizontal beam.

The Normal Pneumoarthrograms: The suprapatellar bursa, the infrapatellar fat pad, the medial meniscus (semilunar cartilage), the lateral meniscus and the popliteal bursa can be clearly delineated. The other structure which can be detected fairly well, but not sufficiently accurately for diagnosis are: the synovial reflection over the popliteus muscle; the crucial ligaments; the articular cartilages covering the femoral condyles; the tibial condyles and the patella; the posterior septum. The presence of fluid in the joint space may interfere. It is best to remove as much as possible or wait for its absorption before performing the examination. There is a complete description of the normal and abnormal variations which have been observed.

The analysis of the results is divided into two periods. In the first between April, 1942 and April, 1944, 528 air injections were done and there were 230 arthrotomies. In the second period from April, 1944 to April, 1945, 254 air injections were done and 85 arthrotomies performed. Because of experience in interpretation and improvement in techniques the accuracy of diagnosis rose from 64.8 per cent in the first period to 81.6 per cent in the second period. Of equal importance is the fact that with experience the number of unsatisfactory examinations decreased from 14.5 per cent to 2.3 per cent. The mistaken diagnoses only fell from 20.7 per cent to 16.1 per cent. It is stressed that the improvement in the accuracy of diagnosis was due to a lessening of the errors of omission rather than of commission.

Abnormalities of the lateral meniscus and medial meniscus are found in a ratio of 2 to 5 and together amount to two-thirds of the major lesions in internal derangement of the knee joint. The other lesions found are abnormal fat pads in somewhat less than one-third of the cases. As a rule, fat pad abnormalities are secondary to some other pathologic process in the joint.

About 25 per cent of the patients had osteochondritis dissecans or chondrosis of the patella. This frequency is one-half that of the medial meniscus lesions and 5 per cent greater than lateral meniscus lesions. Twenty-five per cent of the cases with osteochondritis or chondrosis had loose bodies in the joint; in about half there were other pathologic changes such as fractured menisci.

Popliteal bursae or cysts were present in 13.5 per cent of the cases and are probably without pathological significance unless associated with inflammatory changes or if the communication with the joint is interfered with. Cornu fractures of a meniscus are very difficult to diagnose and may escape detection.

In the present stage of development, the pneumoarthrogram is 80 per cent accurate. The authors believe that the clinical examination at best is approximately 70 per cent accurate. In the case of the pneumoarthrogram, the type and completeness of the lesion can be more accurately predicted. This is especially valuable when both the medial and lateral menisci are involved in the same knee.

The air study and the clinical examination are frequently complementary and permit a greater accuracy when employed together. With both studies an accuracy of diagnosis in the neighborhood of 90 per cent should be achieved. —*Norman Heilbrun, M.D.*

Jacobs, Lewis G. Isolated fracture of the pisiform bone. *Radiology*, April, 1948, *50*, 529–531.

There are only 18 examples of isolated pisiform fracture on record. Incidence estimated not to exceed 1 case in 100 fractures of carpal bones exclusively. Dislocation of pisiform reported with about the same frequency.

Author describes position for proper roentgenogram and discusses mechanism for cause of fracture.

Clinical history of author's patient is briefly described. Roentgenograms of the fractured pisiform are presented.—*E. E. Seedorf, M.D.*

Coles, William C., and Schultz, Milford D. Bone involvement in malignant lymphoma. *Radiology*, April, 1948, *50*, 458–462.

Review of 288 histopathologically proved cases of malignant lymphoma to determine the percentage having bone involvement and determine what influence such involvement may have on the course of the disease or well-being of the patient.

Determination of bone involvement was made by roentgenograms and postmortem examination. Thirty-eight, or 13 per cent, of the 288 patients had manifest involvement of bone at time of first observation or sometime later in the course of the disease. Giant follicular lymphoma was found to involve the skeletal system in a disproportionally low degree while reticulum cell sarcoma revealed skeletal involvement in a rather high degree.

Fifteen patients had solitary involvement and in all there were 113 separate areas of bone involvement in the 38 patients. The spine is the most frequently affected.

Age range of patients was from seven to eighty-three years and ratio of male to female patients, 1.3 to 1. No significant age or sex predilection for bone involvement in malignant lymphoma was found.

With exception of solitary osseous involvement by reticulum cell sarcoma where the object is to try to cure, the attitude in treatment of bone lesions has been essentially that entertained in treatment of malignant lymphoma in general, that is, an attempt at palliation, the intent being to relieve symptoms as they appear. The amount of radiation varies from 600 to 1,500 r.

With the exception of solitary reticulum cell sarcoma of bone, which may be a curable disease, the appearance of bone involvement seems to have no noticeable effect on the survival rate. Neither does the time of appearance of the bone lesion, whether early or late in the course of the disease affect appreciably the ultimate prognosis, except in widespread reticulum cell sarcoma when it tends to occur terminally.—*Robert D. Moreton, M.D.*

Platt, Joseph L., and Eisenberg, Richard B. Eosinophilic granuloma of bone; report of six cases. *J. Bone & Joint Surg.*, July, 1948, *30-A*, 761–768.

In this paper 5 cases of eosinophilic granuloma of bone are summarized. A sixth case is presented in more detail as it illustrates the benign nature of the lesion and the results of conservative treatment.

Laboratory studies, although not complete in every case, were not helpful in making a diagnosis. The authors believe the actual diagnosis rests upon histopathological study. In the discussion of therapeusis, the strong tendency to healing of the lesion is emphasized. This is apparent in view of the uniformly good results following a variety of therapeutic measures. Beneficial results were obtained from roentgen therapy. The roentgen technique used varied for the 6 cases, in some cases 200 kv. was employed, in others 135 kv. The dosage at each treatment varied from 50 to 200 r, the time interval being daily doses or on alternate days. The total dosage ranged from 300 r to 900 r. Wide surgical excision, which was carried out in one case, now seems to the authors not justified. Their experience as carried out in this case, together with the information gained from an analysis of the reported cases, suggests that surgery in suspected cases of eosinophilic granuloma of bone might well be limited to that necessary to secure an adequate biopsy or to relieve local pressure in skull lesions, and that, after the diagnosis has been established, this should be followed by radiation treatment of all the lesions.—*R. S. Bromer, M. D.*

Babaïantz, Léon. Les ostéopathies atrophiques. (Atrophic osteopathies.) *J. de radiol. et d'électrol.*, No. 7–8, 1948, *29*, 333–362.

In these 29 pages every aspect of atrophic osteopathies is discussed and the thoroughness of the review leaves nothing to be desired. The English Summary at the close of the article is quoted in full:

"The author studies atrophic osteopathies (A.O.), for the most part metabolic, in the light of the latest scientific acquirements in histobiochemistry and radiologic morphology.

"Knowledge of physiopathic conditions and histogenetic mechanisms is essential for the understanding and distinguishing of the various kinds of osteopathies, especially as regards their relation with phosphocalcic or proteic metabolism disturbances, and with other endogenous and exogenous factors.

"Bone decalcification must be of some im-

portance—about 50 per cent—to be visible on the X-ray film, as the author has shown in his experiments with chemically decalcified bones.

"Statement of the radiological semiology of A. O. Classification of osteopathies according to their clinical particularities in 8 groups with statement of the histogenetic mechanisms and the essential biochemical data particular to each group. 1. Senile osteoporosis; 2. Deficiency osteopathies; 3. Hormonal osteopathies; 4. Splanchnic osteopathies; 5. Toxic osteopathies; 6. Myelogenic osteopathies; 7. 'Thésaurismoses'; 8. Idiopathic osteopathies.

"The radiological finding of a bony atrophy often reveals an unknown disturbance of protein or mineral metabolism. This must be looked our for. Morphological diagnosis of A.O. by X-rays is only possible in a limited number of bone dystrophies showing a typical radiological picture. Generally only a bone biopsy can offer certainty. Etiology, as a basis for a rational treatment, will be established by anamnestic, radioclinical and biochemical data."—*William M. Loehr, M.D.*

Marques, P., Grimoud, M., and Pouch, G. La dégénérescence maligne des tumeurs a myéloplaxes. (Malignant degeneration of giant cell tumors of bone.) *J. de radiol. et d'électrol.*, No. 3–4, 1948, *29*, 116–119.

Five cases of malignant changes occurring in giant cell tumor of bone are reported and the authors discuss the present ideas concerning such changes in benign giant cell tumor. As for the possibility of therapy being a cause of malignant change in these tumors, the authors state that one should not forget that in the majority of cases these same therapeutic measures bring about healing and one cannot, therefore, ascribe to therapy the sole factor of cancerization. The authors insist on the possibility of malignant degeneration of giant cell tumors of bone. This transformation (quite rare) cannot be forseen in any particular case. At the most, certain forms showing rapid progress and development can only be suspected of malignant potentialities and treated as a malignant tumor whether by surgery or radiation therapy.—*William M. Loehr, M.D.*

Sherman, Robert S., and Pearson, T. Arthur. The roentgenographic appearance of renal cancer metastasis in bone. *Cancer*, July, 1948, *1*, 276–285.

A study of 36 proved cases of renal metastases in bone with adequate roentgenographic coverage was made to determine if these lesions might present characteristics to differentiate them from any other form of skeletal carcinomatous metastases. No correlation between the cell type and the roentgenographic appearance of the metastases was evident.

In no instance was the original roentgenographic diagnosis of renal cancer metastases made on the basis of the roentgenographic appearances alone. Eighteen cases were diagnosed as metastases, however. Four cases were called osteogenic sarcoma, and in one, an amputation was done for the mistaken diagnosis of primary bone tumor. The high percentage of solitary metastases of slow growth enhances this difficulty.

Twenty-one of the 36 cases were solitary. In 15 instances where two or more tumors were seen, the appearance of the lesions was fundamentally alike. No significant site of predilection to certain bones was evident. In 34 of the 36 cases, the medulla was thought to be the site of origin. Some of the tumors showed an early tendency to extend outward beyond the bone into the soft parts. It seems that an oval shape is the more characteristic of renal bone metastasis. Destruction of bone was a constant finding, yet productive changes of a lesser degree were found in 16 cases. Periosteal reaction was seen in 5 instances; cortical destruction with the formation of a periosseous mass was common. Pathological fractures occurred in half of the cases.

Analysis of the roentgenographic picture indicates three types of renal metastases: the lytic, the septate, and the patchy. The lytic was most common (27 cases); the septate was found in 6 cases; and the patchy in 3 cases. There is some tendency toward septa formation in the lytic type especially following roentgen therapy. Recognition of the septate form roentgenographically offers the possibility of diagnosing these lesions as cancer metastases of renal origin.—*J. A. Campbell, M.D.*

Bickel, William H., Young, H. Herman, Pfuetze, Karl H., and Norley, Theodore. Streptomycin in tuberculosis of bone and joint. *J.A.M.A.*, June 19, 1948, *137*, 682–687.

Sixteen patients with tuberculosis of bones or joints were treated using streptomycin in addition to the usual treatment in such conditions.

The authors believe 1 gm. of streptomycin each day given intramuscularly every eight to

twelve hours to be the optimal dose. This is continued for 90 to 120 days.

In this series of cases the treatment with streptomycin varied between 66 days and 190 days with an average duration of 112 days. The average total dose of streptomycin was 134 gm. The daily intramuscular dose of streptomycin varied from 0.75 to 2 gm.

Mild to moderate toxic symptoms were found in 9 of the 16 patients. One patient required discontinuation of the drug. This patient, as well as the others, was desensitized and the drug was continued as previously planned.

A detailed report is given of each case. Response to streptomycin was considered favorable in 9 cases and fair in 1 case; there was no benefit in 4 cases, and the therapy was too recent to evaluate in 2 cases.—*V. L. Peterson, M.D.*

Jaffe, Henry L., and Lichtenstein, Louis. Chondromyxoid fibroma of bone. *Arch. Path.*, April, 1948, *45*, 541–551.

The authors present a series of 8 cases of a rare and peculiar benign connective tissue tumor of bone which they feel must be regarded as a pathological entity. The tumor is characterized pathologically by a mixture of chondroid and myxoid elements and clinically by slow evolution and a benign course. The danger is that it may be misdiagnosed a sarcoma and thus overtreated.

There is no particular age or sex predilection. All of the eight tumors were in the femur, tibia or some bone of the foot. Those in the femur and tibia were in the metaphyses adjacent to the knee, eccentrically placed, eroding the cortex but never piercing the periosteum. There is often a local palpable bulge. They were often bounded inwardly by a zone of sclerosed bone and outwardly by a thin shell of subperiosteal bone. The article includes several roentgenographic illustrations of these features. Among lesions which may be confused with these tumors are bone cysts, enchondromas and foci of fibrous dysplasia.

Treatment has been by curettage, and none have recurred as yet (1½ to 7 year follow-up in 6 cases).—*D. D. Beiler, M.D., and C. L. Hinkel, M.D.*

Toone, Elam C., Jr. Rheumatoid spondylitis: Observations on the incidence and response to therapy among veterans of the recent war. *Ann. Int. Med.*, April, 1949, *30*, 733–739.

This report deals with a series of 29 cases of rheumatoid spondylitis. During the period under consideration there were 8,937 admissions to the hospital as a whole and 197 admissions to the wards set aside for the care of arthritic patients. Rheumatoid spondylitis is a disease of unknown etiology capable of producing severe crippling deformities and is particularly prone to affect otherwise healthy young males. Information from reports indicate that its incidence is increasing. Precipitating factors such as trauma, exposure, infection and climatic conditions are variable and inconstant, and knowledge about the cause and pathogenesis of the disease is inadequate. Roentgen therapy is a valuable means of controlling pain and relieving muscle spasm, but must be combined with measures of a general supportive nature and with measures designed to prevent or correct deformities.

Pain is the universal complaint of patients with rheumatoid spondylitis and so was found to be the major complaint in each of the patients of this series. At the onset the pain is usually localized in the lower back, is often sharp and stabbing in character, intermittent in occurrence and tending to be more severe at night. The average weight loss in this group was 18 pounds with variation from no loss to a loss of 46 pounds. Discouragement, frustration, and severe mental depression were frequently encountered. Almost all of the patients felt discouraged and confused by previous varied diagnoses and inappropriate and ineffective management. Only 2 patients had previously received roentgen therapy.

Roentgenographic changes are probably the most valuable single finding in the diagnosis of rheumatoid spondylitis. The sacroiliac joints are the most important joints to be examined and will almost always show a bilateral involvement if the disease is present. This may vary from an early narrowing of the joint space with irregular bone proliferation and osteoporosis of the adjacent bone to complete ankylosis. Each of the 29 cases in this series showed bilateral involvement of these joints. The apophyseal joints are more difficult to visualize and special oblique views are necessary to demonstrate any change. Here again changes vary from narrowing of the joint space to complete ankylosis. Calcification of the anterior and lateral ligaments and of the ligamentum flavum is a common finding and may appear relatively early in the disease. Fifteen cases showed evidence of calcification in one or more of these ligaments.

Treatments are given at biweekly intervals for two weeks until each area selected for treatment has received 600 r and the patient is allowed a rest interval of from six to eight weeks and the course is repeated. The average patient receives a total of three courses in this manner and then further treatment is withheld for six months unless a relapse occurs. At the end of six months the patient returns for re-evaluation and treatment is repeated or withheld according to his response. The mode of action of roentgen rays in relieving pain in rheumatoid spondylitis is not understood, but apparently its greatest beneficial effect results from its ability to lessen muscle spasm.—*Eugene J. McDonald, M.D.*

Wertheimer, Luiz Gustavo. Coracoclavicular joint; surgical treatment of a painful syndrome caused by an anomalous joint. *J. Bone & Joint Surg.*, July, 1948, *30-A*, 570–578.

In the normal human skeleton, the bones of the shoulder girdle are joined together by the acromioclavicular articulation and by the trapezoid and conoid ligaments, which go from the inferior surface of the lateral third of the clavicle to the body of the coracoid process. These coracoclavicular ligaments help to unite the clavicle and scapula and, with their form and elasticity, they allow a certain degree of dislocation between these bones in movements that have as centers the acromioclavicular and sternoclavicular articulations. Occasionally however, there is found an articular formation which joins the two bones of the scapular girdle, substituting for the trapezoid and conoid ligaments the "articulus coracoclavicularis."

Comparatively few cases of this condition have been reported in the literature. The known total to date is 50 cases. In one reported case, a woman who had fractured the surgical necks of both humeri and who had bilateral coracoclavicular joints, the articulations were regarded as predisposing factors in the production of the fractures. Some writers have attributed pain and limitation of shoulder movements to these anomalous joints. In another case a painful cervicobrachial syndrome improved after resection of a coracoclavicular joint.

The author's patient was a Negro, male, aged thirty-seven, who complained of pain in the shoulder for the last three years before admission to the hospital. The pain, following a comparatively slight trauma, was continuous, radiating to the arm, and increased with exercise and persisted during rest. The roentgenograms of the shoulder showed no local alteration, except for an anomalous coracoclavicular joint; a bony projection, 1 cm. long, arose from the site of union of the middle and lateral thirds of the clavicle and was directed downward and outward. It was slightly enlarged at its free end which was directed downward and inward. At the base of the coracoid process was a formation directed toward the clavicular process, with its free surface adjusted exactly, and from which it was separated by a space of 3 mm. Roentgenograms of the opposite side showed no similar anomaly. Operation was performed, resection of the coracoclavicular joint, following which the patient improved. Three months after operation he was free from pain, movements were not restricted, and it was possible for him to join his hands over his head with his arm fully extended.

The author states the essential point is to demonstrate the existence of an articulation, whether it be a diarthrosis, a symphysis or another type of joint. Schlyvitch, quoted by the author, stated roentgenograms are open to question and that a true articulation can only be shown by dissection. Other authors have asserted that the presence of a clavicular bony process with a facet adapted to the adjacent coracoid process, like the articular surfaces of other true articulations and with a clear articular fissure, leaves no doubt as to the existence of other elements, such as an articular capsule, which cannot be demonstrated in a roentgenogram.

In his first case, no other cause of pain could be found and only the anomalous joint could be held responsible. It is possible that this joint hindered the free motion of the shoulder, especially when the patient's work involved heavy labor. It is questionable whether, as in some reported cases, osteoarthritis of the shoulder may later develop. His second case was a chance discovery of the anomalous joint in a patient suffering from thoracic contusion. There were no complaints that could be attributed to its presence. The roentgenogram showed the abnormal articulation but not as well developed as in his first patient.—*R. S. Bromer, M.D.*

Arkin, Alvin M., and Schein, Albert J. Aseptic necrosis in Gaucher's disease. *J. Bone & Joint Surg.*, July, 1948, *30-A*, 631–641.

In a previous paper, the authors described and attempted to classify hip-joint changes in

Gaucher's disease. Apart from the statement that changes in the femoral head in children resemble those of Legg-Perthes disease, and that the ultimate changes in the hip-joint in adults might be of degenerative origin and might be secondary to these childhood lesions, no attempt was made to interpret the pathological-physiological background for these changes. Subsequently, in one patient treated by prolonged relief from weight-bearing, almost complete restitution of the femoral head took place. At about the same time, cases of Gaucher's disease in adults were encountered, which markedly resembled cases of caisson disease, at least with regard to the bone changes. In addition, in one of their adult cases, roentgenograms taken during childhood indicated that the condition actually had begun, as they had hypothesized, in childhood as a Legg-Perthes-like condition. They present this paper for the purpose of drawing together these various observations, as they are related to the occurrence of a pattern which suggests that aseptic necrosis of bone is a significant factor in Gaucher's disease of bone.

Phemister discussed the known and unknown causes of aseptic necrosis in the femoral head. He included the lesions of traumatic origin, such as fractures and dislocations involving the known blood supply of the injured parts, and non-traumatic lesions, such as caisson disease, arteriosclerosis, osteochondritis dissecans of the hip in adults, and Legg-Perthes disease. He did not mention Gaucher's disease except to state that he had seen collapse of the femoral head with osteo-arthritis of the hip in Gaucher's disease, which resembled idiopathic osteochondritis dissecans of the hip in adults.

Six cases are reported with roentgenographic studies. The first case, first seen when the patient, a girl, was ten years old, is the only case report Arkin and Schein could find in which reconstitution to a state approximating normal, roentgenographically and clinically, occurred in a case of Gaucher's disease involving the femoral head and hip-joint, while the patient was under observation. Presumably, this fortunate outcome was due, as is true in Legg-Perthes disease, to protection from weight-bearing while the necrotic-appearing, reorganized bone and cartilage of the involved joint were still in the plastic stage. They state it remains to be seen whether further changes during late adolescence and adult life will ensue, including hypertrophic osteoarthritis. In the second case, in an older patient previously reported, roentgenograms have lately been found in the library of the X-ray Department which are the original roentgenograms taken during adolescence showing an earlier condition of the hip joint which resembles the infiltrative, Legg-Perthes-like progress seen in the first case. In the third case, a male aged thirty-nine, the changes in the hips suggest the end stages of advanced aseptic necrosis of both femoral heads. In the fourth case, a male, aged fifty, the changes in the hip resemble those found in caisson disease but a routine sternal bone marrow puncture showed the basic nature of the condition was Gaucher's disease. In the fifth case, roentgenograms of the left femur showed typical changes of Gaucher's disease. In the upper portion of the left tibia, a mild sclerosing lesion of bone was present in the metaphyseal region, reminiscent of absorbing caisson disease or aseptic infarction of bone. The same type of lesion was found in the sixth case.

In their discussion of the pathogenesis of the disease, the authors believe that the most likely explanation of their findings is that Gaucher cells, which are known to surround and infiltrate small arteries and capillaries, at times sufficiently cut off circulation to produce aseptic necrosis of bone. Roentgenographically and clinically, the course parallels that of aseptic necrosis of other etiology in which the pathology has been confirmed.—*R. S. Bromer, M.D.*

Howorth, M. Beckett. Coxa plana. *J. Bone & Joint Surg.*, July, 1948, *30-A*, 601–620.

The basis of this report is a study of 115 cases of coxa plana, seen in various stages of the disease, during the ten years from 1933 to 1944 inclusive. In it, the clinical features, the etiology, pathological findings, stages of the disease, laboratory findings, roentgenographic features, differential diagnosis and treatment are discussed.

Howorth does not regard the name coxa plana as altogether satisfactory, but it is the best name so far suggested. The condition is also known as Legg-Perthes disease or Calve's osteochondritis deformans juvenilis, and aseptic necrosis of the upper femoral epiphysis. He believes the term aseptic necrosis is not accurate for describing either the pathological or roentgenographic features of the disease, and should be discarded. Coxa plana, he states, is one of a group of diseases, the osteochondroses, which include slipping of the upper femoral

epiphysis, coxa magna, Osgood-Schlatter disease, apophysitis of the calcaneus, Köhler's disease, Freiberg's disease, Kienböck's disease and epiphysitis of the spine (Scheuermann's disease). They are all characterized by degenerative changes, due to circulatory disturbance, but occur at various ages in various epiphyses or small bones. The age differences of the osteochondroses are explained by the varying vulnerability of the local circulation at different ages. Degenerative changes similar to those of coxa plana may be produced, however, by circulatory damage at other ages, for example, after slipping of the upper femoral epiphysis or fracture of the femoral neck.

The pathological changes in coxa plana were studied at operation in 50 cases. They are summarized as follows: *Early Stage:* synovial membrane swollen, edematous; lymphocytic infiltration, especially about the blood vessels; scattered plasma cells and mononuclear wandering cells (histiocytes); degeneration of hyaline cartilage of the epiphyseal plate; fibrocartilaginous repair at junction of plate with neck; irregular architecture of bony trabeculae of capital epiphysis. *Late Changes:* synovial membrane thickened, avascular, and sclerotic; capsule thickened and contracted.

The first evidence of the disease in the roentgenograms is a globular swelling of the capsule. The joint space appears wider medially, because of a slight lateral subluxation. It appears wider superiorly, because of a flattening of the ossified portion of the epiphysis; but there is no change in the shape of the articular cartilage at this stage.

Soon after the onset, the epiphyseal line appears wider and is irregularly decalcified at its junction with the neck. An irregular dense area soon appears in the capital epiphysis, usually in the central portion. Gradually the dense area becomes irregularly decalcified, but it may enlarge at the same time. During the decalcification, the head becomes wider, flatter and thinner, and the neck becomes thicker and shorter. Gradually the soft tissue swelling subsides. The epiphyseal line recalcifies, while the proximal surface of the neck becomes convex and grows into the head. More gradually the decalcified areas are recalcified. Eventually normal trabeculation occurs, but the head retains its mushroom shape and tends to remain subluxated. The shape of the acetabulum usually follows somewhat the shape of the head. Osteo-arthritic changes often develop in middle life, but these are usually not so severe as those found in cases with slipping of the upper femoral epiphysis, probably because of greater mechanical and physiological disturbance later in life.

Degenerative changes, similar to those in coxa plana, often occur in the course of treatment of congenital dislocation of the hip, slipping of the upper femoral epiphysis, or fracture of the neck of the femur. These changes are due to interference with the circulation to the head, either by "wringing out" of the blood vessels along the neck by immobilization in full extension often with abduction and internal rotation, or by separation of the head from the neck in the course of treatment. Similar degenerative changes occur in suppurative arthritis of the hip due to inflammatory and mechanical pressure interference with circulation to the femoral head.

Coxa plana runs a self-limited course over a period of years and always heals. There is always a residual deformity. The head and neck are broad, the head is flattened and shallow and the acetabulum is somewhat flattened and shallow to correspond to the head. Treatment has little effect on the course of the disease. Bed rest is the treatment of choice in the incipient or acute and rapidly progressive phases of the disease. The hip should be kept slightly flexed for relaxation of the soft tissues and improvement in circulation. The best ambulatory treatment is the suspension of the extremity from a sling and belt with the use of crutches. This should be continued while there is pain and spasm and while the head is soft. The drilling operation, when done skillfully and gently, with a minimum of damage to the circulation, results in more rapid healing of the lesion with less deformity, but the period of healing is still much too long.—*R. S. Bromer, M.D.*

Ghormley, Ralph K., and Clegg, Reed S. Bone and joint changes in hemophilia; with report of cases of so-called hemophilic pseudotumor. *J. Bone & Joint Surg.*, July, 1948, *30-A*, 589–600.

The changes seen in the joints of persons affected by hemophilia are familiar to members of the medical profession. In addition, changes may be seen in the shafts of the long bones in some cases of hemophilia. Such changes are seen much less frequently than are the joint changes and, in the experience of the authors

of this paper, are more severe and result in disaster in most instances. In their series of 44 patients in whom pathological changes developed in the bones and joints, all were males. Six patients were included in the series, 5 of whom are known to have hemophilia; 1 had some type of blood dyscrasia or hemorrhagic diathesis which was never positively diagnosed. The extensive changes found in the joints are familiar to those interested in hemophilia, but their experience has led them to believe that the bone changes, such as those seen in these cases, are much less frequent and are less familiar to all interested in the disease than are the changes in the joints. One of the 6 cases had involvement of the phalanges of the thumb, 1 with involvement of the olecranon and 4 with involvement of the femur. In all cases the most nearly adjacent joint was involved to some extent, and reasonable doubt may exist as to whether the bone changes were secondary to hemorrhages within the joint or whether they were due to primary hemorrhages within the bone or in the subperiosteal tissue adjacent to the bone.

The 6 cases are reported in detail with roentgenograms illustrating the bone lesions. The case with involvement of the phalanges of the thumb showed in the roentgenogram a large tumor-like shadow with destruction of the terminal phalanx. In the cases involving the femur there were varying amounts of destruction of the cortex and in the case involving the olecranon a cyst-like area was present in the olecranon process. Of the 4 patients with involvement of the femur, all are now dead.

Müller, who reported the results of roentgen therapy in 2 cases, is quoted by the authors. He arrived at the conclusion that irradiation had influenced the resorption of the tumor primarily by changing the reaction of the reticulo-endothelial system, as well as by influencing the products of cell destruction. Others have maintained that the roentgen rays influence the constitution of the blood. In the one patient of the authors' series who was treated by roentgen therapy, no improvement was noted.

From the study of the cases reported, Ghormley and Clegg believe it reasonable to conclude that these pseudotumors may arise (1) from hemorrhages originating in the joint, extending along the bone to produce erosion; (2) from subperiosteal hemorrhages which may lead at first to formation of new bone and later to absorption and destruction of bone, and (3) from cortical or medullary hemorrhage, which may lead to cystic changes and later may destroy the bone or lead to fracture and further hemorrhage. It is difficult to state accurately in any one case which route was followed. On the other hand, it seems important to be able to recognize the condition early enough so that destructive changes may be kept under control.—*R. S. Bromer, M.D.*

Horwitz, Thomas. Bone and cartilage debris in the synovial membrane; its significance in the early diagnosis of neuro-arthropathy. *J. Bone & Joint Surg.*, July, 1948 *30-A*, 579–588.

This paper is devoted to the significance of the anatomical finding of bone and cartilage debris (detritus) in the synovial membrane as a possible indicator of the early evolutionary stage of a neuropathy of a joint, notably in cases in which the clinical and roentgenographic findings, at least as far as the joints are concerned, are still those of an osteoarthritis. Attention is paid specifically to 5 cases with involvement of the knee joint. In 3 of these, the diagnosis of neuro-arthropathy was first suggested by the results of histopathological examination of excised synovial membrane. In the other 2, while there was some clinical evidence in favor of neuro-arthropathy, the diagnosis was substantiated by the pathological findings.

Horwitz concludes that the observation of bone and cartilage debris ground into the synovial membrane appears to have diagnostic value in the early evolutionary stages of neuro-arthropathy in cases wherein the clinical and roentgenographic features are equivocal, being still those of an osteoarthritis. It has been a constant finding in the pathological material from all patients with neuropathic joints. Furthermore, the amount of bone and cartilage detritus appeared proportionate to the degree of disruption of the remaining components of the affected joint, and to the stage of evolution of the neuro-arthropathy and of its underlying neurological disease. However, he found that in a control group of 30 cases with operable lesions of the knee joint, it was also found in the material from 2 cases of advanced degenerative arthritis of the knee joint without a neurogenic basis. Therefore, he observes that the presence of imbedded debris, while highly

suggestive, is not altogether specific in the neuro-arthropathies.

The pathogenesis appears to be based on the formation of debris through the erosion of articular cartilage and subchondral bone, which has become ground into the articular soft tissues by continued weight-bearing on a joint less sensitive than a normal joint.—*R. S. Bromer, M.D.*

Grant, Francis C.; Austin, George; Friedenber, Zachary, and Hansen, Alton. A correlation of neurologic, orthopedic and roentgenographic findings in displaced intervertebral discs. *Surg., Gynec. & Obst.*, Nov., 1948, *87*, 561–568.

The purpose of this paper is to present a group of postoperative disc patients who have been critically studied neurologically and orthopedically. The results of these examinations were then integrated with the roentgen findings. In this way the authors hoped to determine any relationship that might exist between the preoperative and postoperative roentgenograms, the operative findings, and the eventual operative result. The summary of their findings follows:

1. A group of 95 postoperative disc patients was studied.
2. Sixty per cent of these patients were regarded as cured and were able to pursue a normal full day's work without any back or sciatic pain.
3. Eighty-seven per cent of these patients were fully satisfied with the results of the operation.
4. The factors of age, trauma, or type of occupation appear to have no relationship to this type of injury. Also the type of operation appeared to be unrelated to the result.
5. Air myelograms while helpful were not as accurate as desired in the author's experience. Oil myelography, preferably with pantopaque as the contrast medium, is the method of choice and should be routinely employed.
6. The end-results will be more gratifying in proportion to the degree of displacement of the nucleus pulposus.
7. Lost or diminished achilles reflexes do not often return, nor should a patient be allowed to believe paralysis will invariably recover following successful removal of the offending mass.
8. No relationship between increased narrowing of an intervertebral space and clinical result can be shown. Fusion of the vertebral bodies or dense bridging occurred in 6 patients, all of whom are in the group of clinically well. It could be postulated that more will develop this change as time goes on, but further follow-up studies will be necessary to substantiate or disprove this point.
9. It seems unlikely that the presence of a significant quantity of residual opaque oil in the caudal sac has any influence upon the clinical result.
10. The fundamental principle for good results is careful selection of patients with exclusion of those cases that fail to measure up to an exacting history and physical examination supported by myelography.—*Mary Frances Vastine, M.D.*

Bartelink, D. L. Myelography in intervertebral disc protrusion; horizontal beam examination with the patient prone. *Radiology*, Feb., 1948, *50*, 202–206.

A narrow dural sac and lateral protrusion of the intervertebral disc are not the only reasons for failure of operatively proved disc hernias to produce a characteristic defect in the contrast oil column. The author demonstrates some anatomic reasons for certain shortcomings of conventional vertical beam myelography, and describes a method of eliminating some possible false negative diagnoses of lumbar intervertebral disc protrusion. The prone patient is examined on a special narrow tilt table placed between the table and screen of the vertical fluoroscope, using maximum milliamperage, high kilovoltage, and a fine grid.

The dural sac was not found in direct contact with the posterior surfaces of the vertebral bodies and discs in 15 per cent of cases, this epidural space varying from 2 to 10 mm. in depth, and being greatest at the lumbosacral disc level. Hence, in these cases an intervertebral hernia cannot reach the dural sac and produce a defect in the opaque column unless the size of the protrusion be considerable. However, the herniated disc may compress the nerve root passing over it and produce a root sleeve defect in the myelogram in the

absence of defect in the contrast oil column.

Concavity of the posterior surfaces of the vertebral bodies, and bony productive changes at the posterior margins of the vertebral bodies may occur, with or without true disc protrusion, and produce narrowing or even separation of the opaque column as seen on the conventional vertical beam myelogram. By use of horizontal beam myelography, the true disc protrusion is apparent as a definite defect in the ventral surface of the column at the disc level, whereas no such defect is seen when only vertebral anatomic variations exist without concomitant disc protrusion.

The horizontal beam prone examination is also useful when faulty injection results in the deposit of opaque medium both intradural and extradural. On lateral fluoroscopy the intradural deposit is seen ventral to the epidural deposit, and can be adequately used for diagnostic myelographic examination.—*Richard E. Lukes, M.D.*

Albertengo, Juan B. Diagnóstico y tratamiento de la hernia del disco intervertebral (lumbar). (Diagnosis and treatment of lumbar intervertebral disc hernia.) *An. de cir.*, June, 1948, *13*, 63–110.

The author presents a detailed study, quoting 134 references, of the entire problem of the herniated lumbar disc. There is discussion of the history of the problem, anatomy, physiology, pathology, symptomatology, clinical history, physical and neurological examination, localization of the lesion, and differential diagnosis. Stress is laid on the importance of evanescence of the symptoms, and their aggravation by coughing and sneezing.

Among supplementary examinations are listed plain roentgenography and myelography. The indications for myelography are discussed, the author believing that myelography is only necessary in atypical and confusing cases, that in the majority of cases correlation of significant changes in the plain films with history and physical findings is sufficient for accurate diagnosis. Present-day myelographic technique is discussed in detail.

There is a long discussion of indications for surgery and of the surgical treatment and the author presents synopses of a series of 10 patients treated by himself, showing varying findings in plain roentgenography and myelography. In a footnote, the author states that he has operated an additional 12 patients successfully.—*William S. Wallace, M.D.*

Guri, J. Puig. Tumors of the vertebral column. *Surg., Gynec. & Obst.*, Nov., 1948, *87*, 583–598.

Abnormalities in the density, structure or general configuration of the vertebral bodies or neural arches may be produced either by tumors arising primarily in the vertebral column or surrounding structures or as a consequence of metastasis from neoplasm located in other parts of the body. In order to obtain a better understanding of these neoplasms the author studied all the patients with tumors of the vertebral column seen at the State University of Iowa Hospitals with good clinical and roentgenographical follow-up.

Hemangioma. Vertebral hemangiomas are found in 10–11 per cent of autopsies. Hemangioma of the cervical vertebrae is a rarity since this condition usually involves the dorsal or lumbar vertebrae. The author reports a case in which the cervical portion of the spine is involved. Roentgen therapy is the treatment of choice unless there are signs of cord compression.

Giant Cell Tumor. These tumors may be osteolytic or osteoblastic. The author presents 2 cases in which the lesion was osteolytic and involved a sacral segment. When these tumors involve only the neural arches or when involvement of the vertebral body is only partial, removal with curettage is probably the treatment of choice. When the tumor involves most of a vertebral body roentgen therapy is indicated. Spontaneous cures may occur following pathological fracture.

Chordoma. Notochordal tissue remnants are found during postmortem examinations in 2 per cent of cases. They usually appear at the base of the skull or in the sacrococcygeal region. On the roentgenogram an area of rarefaction without signs of new bone formation is the usual picture. A soft tissue shadow may also be seen. Total resection is the accepted treatment since these tumors are radioresistant.

Chondroma. Benign cartilaginous tumors arising from the neural arches are frequent while chondromas arising from the vertebral bodies are rare. The prognosis of chondromas is not dependent upon the histopathological picture but upon their localization. When these tumors are localized at the neural arches and grow posteriorly they may reach enormous sizes

without producing serious complications. Treatment is total resection if possible.

Fibrosarcoma. Seven cases of fibrosarcoma were studied. In these 7 the lesion was purely osteolytic. Fullness or presence of a mass in the paravertebral region or on rectal examination was present in 4 cases.

Chondrosarcoma. In the one case studied the chondrosarcoma was localized in the dorsal spine.

Osteogenic sarcoma. In the one case seen by the writer, the tumor was found to involve, the fourth, fifth and sixth cervical vertebrae.

Hodgkin's Disease. In the 4 cases seen the process involved the vertebral bodies and the transverse processes and was osteolytic in character.

Multiple Myeloma. The roentgenographic findings varied from a marked osteoporosis without definite destructive areas to the characteristic multiple and well circumscribed osteolytic lesions.

Solitary Myeloma. Among the 10 cases of multiple myeloma studied by the author there were 2 cases in which the lesion could be considered circumscribed to the spine during the early stages of the disease. In some of the cases of vertebral solitary myeloma reported in the literature, the lesions remained confined to the spine for periods of ten years.

Vertebral Destructions Produced by Metastasis from Primary Tumors Elsewhere in the Body.

1. Carcinoma of the breast.
2. Carcinoma of the prostate.
3. Hypernephroma.
4. Ewing's tumor.
5. Chondrosarcoma.
6. Bronchogenic carcinoma.
7. Carcinoma of the skin.
8. Carcinoma and fibrosarcoma of the cervix.
9. Osteogenic sarcoma.

Vertebral changes from Tumors Arising in the Vertebral Canal. The author found 2 such cases in his material, one a meningioma and the other a perineurial fibroma.—*Mary Frances Vastine, M.D.*

Brav, Ernest A., Molter, Howard A., and Newcomb, Wendell J. The lumbosacral articulation; roentgenologic and clinical study with special reference to narrow disc and lower lumbar displacement. *Surg., Gynec. & Obst.*, Nov., 1948, *87*, 549–559.

In view of the continued difference of opinion relating to the importance of narrowed fifth lumbar disc and displaced fifth lumbar vertebra in the etiology of backache and sciatic pain, a study of a large series of roentgenograms was made with the purpose of establishing the incidence of these conditions.

1. In a series of 500 roentgenograms focused over the lumbosacral joint, narrowed fifth lumbar disc was noted in 26.4 per cent, posterior displacement of the fifth lumbar vertebra was present in 10.2 per cent, anterior displacement of the fifth lumbar was present in 5.0 per cent, and there was displacement of the fourth vertebra in 3.0 per cent.

2. The incidence of back and leg pain in the 181 cases in which these changes were noted was not significantly greater than in the other 319 cases.

3. When the two groups were studied clinically they showed a strikingly similar incidence of alteration in leg reflexes, limitation of straight leg raising, and body tilt on forward bending.

4. The clinical diagnoses in the two groups were almost identical. There was, however, a considerably higher incidence of lumbosacral arthritis in the patients with narrow disc or lower lumbar displacement.

5. A study of the groups of patients with back pain alone, with associated leg pain, and with neither of these complaints revealed no significant difference in the incidence of narrowed fifth lumbar disc. However, there was a smaller incidence of lower lumbar displacement in the symptom-free group.

6. Posterior displacement of the fifth lumbar vertebra is apparently a definite entity and is not due entirely to difference in anteroposterior diameters of the fifth lumbar vertebra and the sacrum. However, in about 20 per cent this is apparently the reason for the appearance on the roentgenogram. It is possible that in cases of posterior displacement, there is secondary atrophy of the anterior edge of the sacrum which decreased the anteroposterior diameter of the first sacral segment.

7. Anterior displacement of the fifth lumbar vertebra is usually associated with a defect in the interarticular portion of this vertebra and in at least 40 per cent of the cases there is anterior lipping of the sacrum.

8. In cases of herniated nucleus pulposus or posterior disc protrusion, the incidence of narrowed fifth lumbar disc or lower lumbar displacement is not significantly greater than in

the remainder of the series. Narrow disc on the roentgenogram cannot be considered clinical evidence of posterior displacement.

9. Narrowed fifth lumbar disc and displacement of the lower lumbar vertebrae are clinically significant only in that these conditions place additional strain on an already mechanically vulnerable lumbosacral joint.

10. The weight of evidence seems to indicate that in most instances narrow fifth lumbar disc and lower lumbar displacement are in themselves not the cause of low back and sciatic pain.—*Mary Frances Vastine, M.D.*

Riker, William, and Potts, Willis J. Sacrococcygeal teratomata in infancy; report of six cases. *Ann. Surg.*, July, 1948, *128*, 89–100.

The authors found an incidence of 1 per cent for sacrococcygeal teratoma of all tumors seen by them in pediatric practice. All 6 of their cases were in females and of those reported in the literature 75 per cent were female. A large percentage are still born or die at birth. All of these cases were less than a year old. The exact etiology is unknown but the two main theories are: (1) the teratoma represents a rudimentary organ mass of an ill developed pygopagus twin, or (2) the tumor arises from cells already in the sacrococcygeal region and its growth includes nervous, intestinal, bony and connective tissue elements. The tumor mass is quite variable in size, usually lobulated with cystic and solid areas containing tissue of any structure in the body. Roentgen examination proves the presence of teeth and malformed bone.

In the differential diagnosis, meningocele can be differentiated from teratoma in that it does not increase in size with the growth of the infant and is covered by a translucent membrane which bulges on crying or coughing. The presence of spina bifida also suggests meningocele. In a review of the literature the authors found a 15 per cent incidence of development of malignancy in sacrococcygeal teratomas. Usually only one tissue element becomes malignant. Complete and early excision of the tumor by the posterior route is emphasized.—*T. D. Allison, M.D., and C. L. Hinkel, M.D.*

Longstreth, H. Paul, Blanco, Pio, and Sanes, Samuel. Myxochondrosarcoma of the talus. *J. Bone & Joint Surg.*, July, 1948, *30-A*, 774–778.

Primary malignant tumors of the talus are rare. In this paper, the authors describe 10 cases collected from the available literature of the past fifty years and they add an additional case of myxochondrosarcoma of the talus in a man, sixty-two years old. The findings of the collected cases and of their patient are given in detail. The latter was the oldest patient in whom this tumor had been described. The tumor was of the central, not the peripheral type. It had extended to the overlying soft parts and formed a fluctuant, pseudocystic, hemorrhagic mass which, with the bone destruction, gave suggestive roentgenographic evidence of the condition. No other patient, reported in the literature, had received roentgen therapy before operation. Whether this treatment had any effect upon inhibiting the growth and spread of the tumor, or upon producing regressive changes in it remains a matter for speculation. The irradiation did not destroy it. In chondrosarcoma of one of the bones of the foot, amputation above the ankle joint is advised, rather than local excision.

The authors' case illustrated that, as in other bones, chondrosarcoma of the talus runs a slow course with metastases, chiefly by way of the veins, to the lungs and the heart. The patient was followed for three years from the onset of symptoms to death from suicide. In no previously reported case of chondrosarcoma of the talus in the literature was an autopsy performed or was the patient proved to have visceral metastases.—*R. S. Bromer, M. D.*

Reich, Carl, and Brodsky, Alexander E. Coexisting multiple myeloma and Paget's disease of bone treated with stilbamidine. *J. Bone & Joint Surg.*, July, 1948, *30-A*, 642–646.

The object of this report as stated by the authors is, first, to describe a case of coexistent multiple myeloma and Paget's disease of bone, and second, to set forth their experiences with stilbamidine in treating this case of multiple myeloma. The diagnosis of multiple myeloma was definitely established in the patient by sternal-marrow studies, but no biopsy material could be obtained from the affected left hip. However the roentgenographic appearance of the femur unquestionably fulfilled the classical criteria of Paget's disease of bone. They are unable to explain the appearance of these two disease entities in their patient, or to postulate any tenable cause-and-effect relationship of the two.

They feel it is important, especially for diagnostic significance and for proper management of the patient, to point out that the two may coexist. Upon looking at the patient's roentgenograms, one might be tempted to explain the appearance on the basis of Paget's disease alone, for the areas of circumscribed osteoporosis, which in this case were caused by multiple myeloma, could have been interpreted as the "osteoporosis circumscripta" of early Paget lesions which they resemble. They believe, also, that their case would indicate the advisability of excluding multiple myeloma, by means of bone marrow studies in those cases of Paget's disease where there are areas of circumscribed osteoporosis, even in the absence of Bence-Jones proteinuria and hyperglobulin-anemia.

Because the roentgenographic appearance of Paget's disease occasionally is simulated by metastases of carcinoma of the prostate, a careful work-up was done to exclude this possibility. No prostatic or other condition to produce this picture was found. The additional possibility of multiple myeloma producing Paget-like changes in the roentgenogram can be ruled out by the absence of any mention of such an occurrence in the numerous reports on multiple myeloma.

Treatment with stilbamidine (a total of 2.85 grams) caused early disappearance of all pain and increased the spinal mobility of the patient. Basophilic granules, which had not been present prior to therapy, appeared in the cytoplasm of the myeloma cells. The patient experienced no serious or permanent ill effects attributable to therapy. The transitory ill effects accompanying the administration of stilbamidine can be avoided by certain simple precautions which they enumerate in the paper. —*R. S. Bromer, M.D.*

BLOOD AND LYMPH SYSTEM

Hoster, H. A., Dratman, M. B., Craver, L. F., and Rolnick, H. A. Hodgkin's disease. *Cancer Research*, Jan.–Feb., 1948, *8*, 1–78.

The purpose of the article is to "stimulate interest and research in Hodgkin's disease and allied fields of investigation." The paper is an extensive review of the literature on all phases of Hodgkin's disease and presents the opinions of many investigators as well as the results of recent scientific investigation. A study as comprehensive as this cannot be summarized in the usual space allotted to an abstract.—*John DeCarlo, Jr., M.D.*

Jackson, Henry, Jr. The practical aspects of the diagnosis, treatment and prognosis of Hodgkin's disease and allied disorders. *Radiology*, April, 1948, *50*, 481–485.

The author, feeling that the term "malignant lymphoma" is too inclusive and actually meaningless, offers a classification which differs only in minor respects from other classification but justifies it on the basis of differences in age, symptoms, therapeutic response and prognosis of various types.

He divides Hodgkin's disease itself into three types: (1) comparatively benign para granuloma, (2) more frequent and fatal granuloma, (3) rare but extremely malignant sarcomatous type.

Giant follicular lymphoma may change to more malignant forms such as Hodgkin's disease and lymphosarcoma. Lymphosarcoma, reticulum cell sarcoma, plasmocytoma and endothelioma are placed in a general group.

The author reviews the relative incidence, ages and symptomatology of the various groups and recommends the types of therapy for each group. Prognosis is summarized for the individual types.—*Robert D. Moreton, M.D.*

Hare, Hugh F., Mulry, William C., and Sornberger, C. Franklin. Lymphoid tumors. *Radiology*, April, 1948, *50*, 506–514.

The authors deplore the defeatist attitude of the general practitioner and specialists toward lymphoid tumors and point out the necessity for a uniform classification.

The classification used in this article is based on the origin of these tumors from a reticulum cell source. From the lymphoid phase of the reticulum cell, lymphosarcoma, lymphocytoma, and lymphatic leukemia are derived; from the reticulum side, Hodgkin's disease develops and from another unclassified section of the reticulum cell, the macrofollicular lymphoma takes it origin.

The authors discuss in detail the etiology, incidence and course of these tumors with the conditions in which a differential diagnosis must be made.

Roentgen therapy as recommended by the authors in both localized and generalized forms of the disease is presented.—*Robert D. Moreton, M.D.*

Moffitt, Herbert C., and Lawrence, John H. Chronic leukemia of long duration; with a report of 31 cases with a duration of over five years. *Ann. Int. Med.*, April, 1949, *30*, 778–790.

The actual disease process may antedate the onset of symptoms by a considerable period. It is well known that chronic leukemia may be detected from routine examination of the blood prior to the onset of any symptoms. Wintrobe and Hasenbush estimate that two to five years—or longer—elapse between the actual time of onset of chronic myelogenous leukemia and the time when symptoms cause the patient to seek medical aid. In chronic lymphatic leukemia they found that one and a half to two and a half years passed between the finding of signs of the disease (leukocytosis, glandular enlargement, or splenomegaly) and the development of symptoms of the disease. They believed lymphatic leukemia was discovered earlier in its course because of the associated lymph node enlargement.

In the course of leukemia, remissions, both symptomatic and hematologic, may occur spontaneously as well as from the effects of treatment. Such spontaneous remissions may contribute to the long duration of many of the unusual cases under discussion but no cause—other than natural variation in the disease—has ever been definitely established for their occurrence.

Various types of infection have been reported as a cause of remission in leukemia. Infection might, therefore, be a factor in cases of long duration. Forkner reviewed the pertinent literature and concluded that, although many miscellaneous infections were associated at times with evidences of regression, in all instances in which recovery from infection occurred leukemic manifestations recurred, usually in a few weeks.

It is generously agreed that there is not yet clean cut evidence that treatment has significantly prolonged the duration of any type of chronic leukemia. However, practically all agree that radiation therapy lengthens the period of comfortable and useful life. The possibility of cure of leukemia is still undetermined. Until more is known about the etiology of leukemia, it cannot be stated definitely whether recovery is possible.

Pascucci summarized the factors which were associated with short duration of the disease in his series of 128 patients from the Presbyterian Hospital in New York. He listed the following findings as indicating a poor prognosis, but stated there were few substantiating statistics: very high or very low white blood cell count, serious anemia, high number of blast cells, excessively high or excessively low platelet count, marked splenomegaly, diffuse lymph node involvement, presence of complications (hydrothorax, pneumonia, tuberculosis, osteomyelitis, cardiovascular disease, etc.) and short duration of symptoms prior to diagnosis.—*Eugene J. McDonald, M.D.*

Farber, S., Diamond, L. K., Mercer, R. D., Sylvester, R. F., and Wolff, J. A. Temporary remissions in acute leukemia in children produced by folic acid antagonist, 4-aminopteroyl-glutamic acid (aminopterin). *New England J. Med.*, June 3, 1948, *238*, 787–793.

The observation by Farber that leukemic children treated with certain folic acid conjugates showed exaggeration or acceleration of the leukemic process suggested that folic acid antagonists might be of value in patients with acute leukemia. After studies with pteroylaspartic acid and methylpteroic acid on 21 patients, a more potent agent, 4-aminopteroylglutamic acid (aminopterin) became available.

In 16 infants and children with acute leukemia treated with aminopterin intramuscularly, 10 showed clinical, hematologic, and pathological evidences of important improvement, although many were moribund at onset of therapy. Six did not respond well; 4 of these are dead, and 2 unimproved. Five of the 10 cases showing significant improvement are presented in detail.

Aminopterin has a marked effect on leukemic bone marrow, on immature cells in the peripheral blood, and very probably on leukemic deposits in the viscera as well. In the treated patients, the white count tended to return to normal, regardless of whether the initial count was abnormally high or indicated a marked leukopenia. The percentage of immature cells fell, and blast forms decreased or disappeared from the peripheral blood. Hemoglobin, red blood cell count, and platelet values approached normal, as did the relative percentage of mature leukocytes in the peripheral blood. Bone marrow studies showed decrease or disappearance of leukemic cells. Enlarged livers of spleens return toward normal size, and adenopathy may disappear.

The authors emphasize that remissions so far have been of short duration, that toxic effects such as stomatitis are already known, that at least 2 of their improved cases had previously shown brief remissions following intercurrent bacteriemias, and that there is as yet no justification for the assumption that a cure for acute leukemia in children has been found.—*Henry P. Brean, M.D.*

Uhlmann, Erich M. The significance of giant follicular lymphadenopathy (Brill-Symmers disease). *Radiology*, Feb., 1948, *50*, 147–156.

Giant follicular lymphadenopathy (Brill-Symmers disease), a condition of rising importance, but little known by the medical profession in general, constitutes a disease entity which may eventually be followed by lymphatic leukemia, polymorphous cell sarcoma, or Hodgkin's disease.

Originally described by Becker in 1901, it was named by Symmers (1927) and is not an uncommon condition.

Characterized by localized or generalized, soft, painless lymphadenopathy, it is often misdiagnosed as Hodgkin's disease. The size, position, or growth rate of the nodes is not regular or characteristic. Splenomegaly may be present, and the peripheral blood is normal. There is no sex preference and the greatest incidence is in the third and fourth decades.

The microscopic picture is characteristic, with change but not destruction of the follicular structure. Often the follicles are filled with large hypochromatic or even achromatic nuclei and the peripheral zone of leukocytes may be absent. Occasionally, the follicles may be made up entirely of small lymphocytes. The change marking the transition to polymorphous cell sarcoma is rupture of the follicles, and invasion of the cells within the surrounding tissues. The nodes may remain static for long periods, but later show transition to one of the more malignant forms.

Briefs of 22 cases with follow-ups varying from one case still under treatment up to a case nine years after treatment, shows 6 deaths; 3 from leukemia, 1 from Hodgkin's disease, 1 from leukosarcomatosis. The author feels that intensive radiation therapy was responsible for the wellbeing of the 9 patients still alive, three to nine years after therapy. Whereas small doses produce regression of the nodes, recurrence is common, and with large doses of radiation, prognosis is better. This condition should be treated from the start as a malignant lesion, not as a benign or a potentially malignant one. This, the author believes, will usually prevent the development of a malignant lymphoma.—*George D. Adams, M.D.*

Calme, A. Application d'une nouvelle substance iodée à l'artériographie et à la phlébographie. (Employment of a new iodized substance in arteriography and phlebography.) *J. de radiol. et d'électrol.*, No. 3–4, 1948, *29*, 101–107.

From the Surgical Clinic of the University of Geneva the author reports the use of a new radiopaque substance in arteriography and phlebography. The report covers 60 arteriograms and phlebograms with the new substance having the trade name Savac. It is chemically a salt of a diethanolamine of a diiodopyridone in 30 per cent solution. One of the illustrations shows a beautiful cerebral arteriogram using 20 cc. of the solution. The material is well tolerated by the patients, the roentgenograms clear and very satisfactory. The material was developed for use in visualization of the circulatory system because the substance of choice (vasoselectan) could no longer be obtained.—*William M. Loehr, M.D.*